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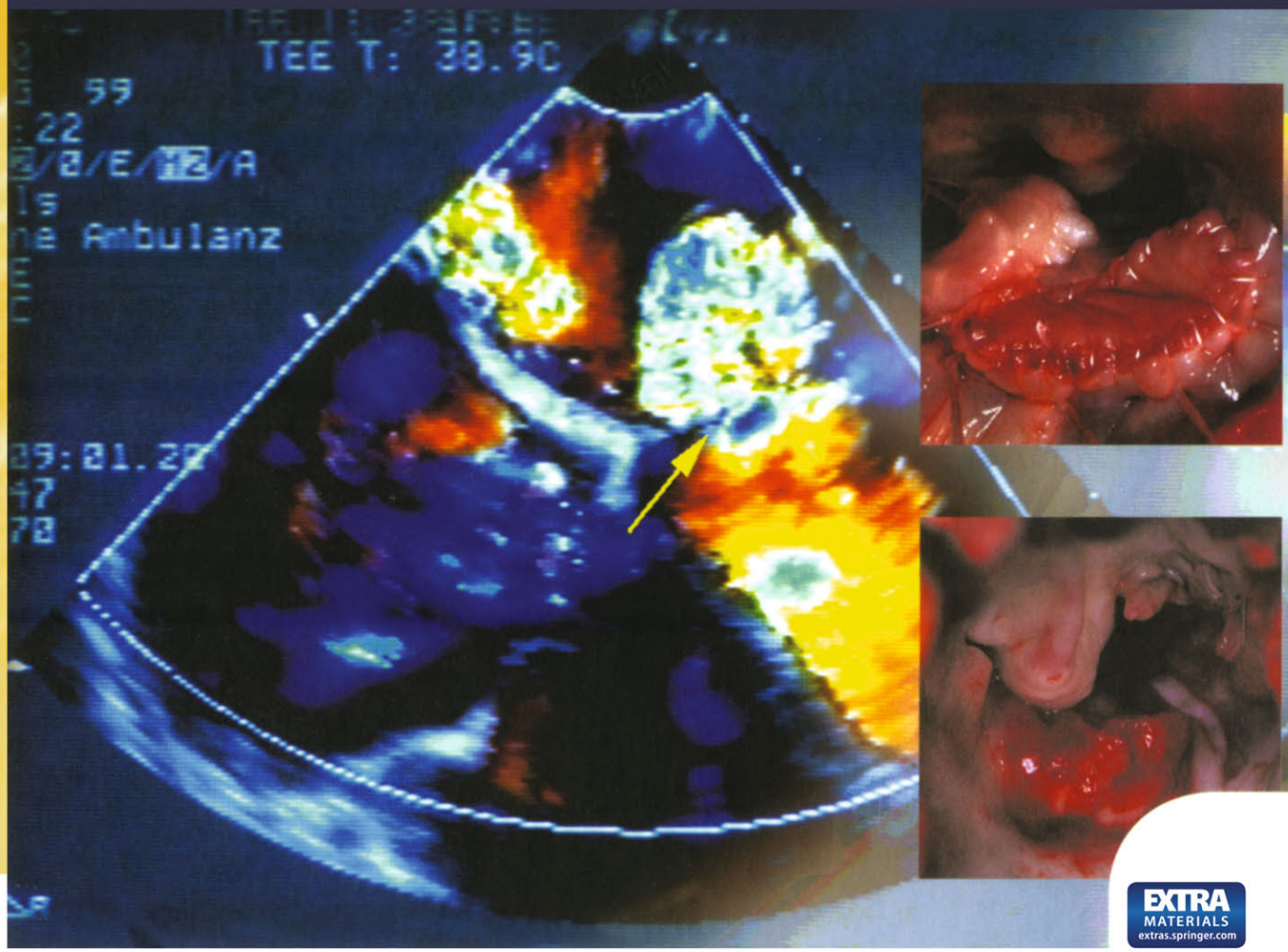

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Modern Mitral Valve Repair

Echocardiographic Interpretations and Surgical Strategies

in collaboration with

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To my parents:
For their love, education and understanding.

And to my former teachers:
Dr. F. Gschnitzer and Dr. M. Turina
who taught me much of what appears
in this book.

飲水思源

"Gratitude for the source of success"
a Chinese proverb

FOREWORD

The magnitude of the problem of mitral regurgitation is clearly in the rise in the western as well in the developing nations. Echocardiography plays a seminal role in the identification and classification of mitral regurgitation as well as the quantification of its severity. Furthermore, echocardiography is the modality of choice for serial assessment of the left ventricular and left atrial remodeling and for noninvasive determination of pulmonary arterial pressures at rest and with exercise in patients with mitral regurgitation. Based on the current quantitative measurements, the presence of severe mitral regurgitations is diagnosed when the regurgitant volume is greater than 60 cc per beat and effective regurgitant orifice is $\geq 0.4\,cm^2$. The presence of quantitatively of severe mitral regurgitation in the context of repairable mitral valve anatomy should be a strong incentive for early mitral valve repair even in asymptomatic patients. This paradigm shift is predicated on the fact that echocardiography in the experienced hands can clearly delineated the morphologic basis of mitral regurgitation and provide excellent assessment of whether the valve is repairable or not. The next step of surgical correction/elimination of mitral regurgitation is even crucially dependent upon the experience and expertise of the surgeon. In this day and age, utilizing the modern techniques, we should expect successful and long-lasting repair being accomplished in greater than 95% of cases of degenerative mitral valve regurgitation, particularly those involving prolapse or ruptured chordal apparatus of myxomatous mitral leaflets. Clearly the prolapsed middle scallop of the posterior mitral leaflet (P2 segment) can be repaired in virtually all cases. In this regards, it is quite disappointing that mitral valve repair is performed in less than half of eligible cases in the United States and statistics are only slightly better for Europe. Therefore, there is a great need for surgeons to learn the art and science of mitral valve repair so that suitable candidates will in fact, have successful and long-lasting mitral valve repair than replacement.

This monograph, which is beautifully illustrated, fills a void that currently exists in correlating echocardiographic, anatomic, and surgical finding in a concise, yet fairly comprehensive book. Modern operative techniques are presented clearly, and while surgeons may debate the relative merits of one method over another, the important anatomical and physiological principles stand out.

We would recommend this text to all clinicians with interest in evaluation and management of mitral regurgitations of different etiologies, and in particular, to those surgeons, cardiologist, and anesthesiologists interested in furthering their understanding of the surgical nuances of mitral repair.

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PREFACE

The knowledge concerning the diagnosis and the treatment of the diseased mitral valve is now in a period of standardized stability. This allows thoughtful evaluation of the principles of diagnosis and treatment as well as the results of operative techniques, which have been used during the previous decade.

Preoperative and intraoperative echocardiography has become an indispensable tool for guiding mitral valve surgery and has fostered development of many innovative surgical techniques. Close cooperation between cardiologists and surgeons is essential, and their roles are complementary as far as the need for surgical intervention and its optimal timing are concerned. The aim of this publication with a DVD included (43 demonstrations for echocardiography and valve repair) is to provide an excellent illustrative guide to teach echocardiographic-anatomic correlations and to educate the readers on various innovative techniques of valvuloplasty. This may narrow the knowledge gap between related specialists; mutual precise interpretations of echocardiography and modern operative strategies thus contribute to achieve favorable results in management of complex mitral valve pathology.

We take great pleasure in expressing our thanks to Dr. Jamil Tajik and Dr. Hartzell V. Schaff for kindly writing the *Foreword* to this book.

I am greatly indebted to the teaching of Prof. Alain Carptentier whose essential techniques I have adopted. A word of gratitude and of respect is due to him.

We wish to express our gratitude for the privilege of editing this work and to thank all the co-authors and colleagues for their unfailing support and their outstanding contributions, especially to Drs. Bernd Eber, Ulli Strasser, Johannes Auer and Herbert Franke. The contributions of Mrs. Marie Hovorkova were crucial in that she performed tasks above and beyond her daily routine responsibilities with a pleasant spirit of cooperation. Mr. Wojciech Karpowicz and Mr. Friedrich Bramböck were equally indispensable as they assisted with data collection and the layout of the photographic illustrations. These illustrations, along with the film footage of actual surgeries, are my own and were recorded for educational purposes. Finally, I owe an immense debt of gratitude to publisher Springer-Verlag Wien New York and their staff for the outstanding work in the production of this book.

All of us would feel amply rewarded and above all justified in the amount of effort and painstaking work demanded of their colleagues, the contributors, if this book will help directly or indirectly some of those patients who suffer from regurgitant disease of the mitral valve.

Wels, July 2003 Choi-Keung Ng

CONTENTS

1 Introduction

Mitral valvuloplasty is an established surgical method for the treatment of mitral insufficiency. Compared to with mitral valve replacement, reconstruction of the mitral valve with preservation of the continuity of the annulus, the chordae tendinae and the papillary muscle is beneficial with improvement of left ventricular function, end-diastolic and end-systolic left ventricular volume and end-systolic left ventricular wall tension. In addition to these surrogate markers, mitral valve repair was shown to reduce mortality in clinical studies. Mitral valvuloplasty is less commonly associated with thromboembolic events, hemorrhagic complications secondary to anticoagulation, hemolysis and endocarditis, and therefore offers definite advantages in comparison with valve replacement [1]–[4].

The success of valvuloplasty is largely dependent on the extent of the underlying disease of the mitral valve. In many cases, prolapse of the posterior mitral leaflet secondary to myxomatous degeneration is treated by quadrangular or triangular resection. However, complex mitral valve disorders secondary to extensive annulus calcification after degenerative processes or rheumatic fever, Barlow's disease, florid destructive endocarditis, ischemic papillary muscle dysfunction, "bi-leaflet" prolapse with multiple lesions or avulsion of both leaflets with involvement of commissures and post-traumatic events are some of the myriad conditions that hinder successful reconstruction of the mitral valve. In such cases, mitral valve replacement is given preference [5], [6].

Failure or inadequate results of surgery have been observed in those cases in which appropriate surgical techniques, intraoperative differentiation of the underlying pathology, or preoperative diagnostic experience in planning repair were lacking. These factors limit adequate valve repair. The correct surgical strategy and selection of the method are dependent on precise conclusions drawn from preoperative echocardiographic investigations [7]–[10].

In order to improve the results of surgery in this patient population, new innovative surgical techniques are required. This book comprises a total of 204 colored illustrations with a DVD, which present preoperative echocardiographic diagnosis, comparison of echocardiographic features with the intraoperative pathology, and valvuloplasty techniques. In different chapters I described my recent experience in reconstruction in patients at hospital Wels, specifically in plastic restoration of valve function by means of endoventricular pericardial patch annuloplasty, rotation paracommissural sliding plasty, leaflet remodeling with leaflet substitute or extension plasty of the posterior or anterior leaflet (or both), sliding plasty with chordal shortening, chordal replacement, leaflet transposition by flip-over of chordae, leaflet sliding plasty, sliding atrioplasty, restoration of the asymmetric geometry of the left ventricle, reimplantation of ruptured papillary muscles in "emergency" indications, and atypical and combined reconstructions. Specific topics such as annulus calcification, Barlow's disease, endocarditis, ischemic disease, trauma, combined defects and multivalvular defects are also discussed here. I hope these case presentations contribute to our goal of successfully repairing severely diseased mitral valves.

2 Classification of mitral insufficiency according to leaflet mobility

The classification selected here is mainly oriented towards *the extent of leaflet mobility*. Leaflet motion may be normal, excessive, or restrictive and this ought be determined before surgery, on the basis of transthoracic echocardiographic (TTE) and transesophageal echocardiographic (TEE) investigations [11]–[14] (Figs. 1, 2).

The mitral valve closing mechanism is complex, and any condition affecting the papillary muscles, chordae tendineae, annulus, leaflets, or the size of the left ventricular cavity could result in a mitral insufficiency. Before attempting to restore normal function to a diseased mitral valve, it is essential to understand the mechanism of valvular dysfunction whereby the numerous pathological processes affecting one or more of the valve components have to be analyzed. For better comprehension and for targeted application of corrective measures, detecting the various forms of mitral insufficiency preoperatively is of decisive importance.

Preoperative evaluation

Echocardiography has the advantage of being noninvasive, enabling serial studies to follow progression of the disease, and is an invaluable adjunct in the decision-making process regarding the appropriate timing for surgical intervention. Echocardiography provides excellent dynamic images of the leaflets, chordae, papillary muscles, and myocardium. Echocardiographic studies demonstrate the mechanism of regurgitation, the quality of mitral valve tissue, and the feasibility of valvuloplasty.

For the future, real time three-dimensional echocardiography may even enhance our abilities to evaluate mitral valve pathology.

Doppler echocardiography is used to determine the severity of mitral regurgitation, and several methods for quantification are in clinical use. The extent to which the systolic flow disturbance extends into the left atrium can be determined with standard pulsed Doppler techniques.

Observation of the direction of the jet by Doppler color-flow imaging is particularly important in determining the mechanism of mitral regurgitation, equally as useful as observing the valvular structure by two-dimensional echocardiography. The direction of the jet is usually opposite the leaflet that is flail or prolapsing: posterior leaflet prolapse or flail causes an anteriorly directed jet; anterior leaflet prolapse or flail causes a posteriorly directed jet.

A central mitral regurgitant jet occurs when the effective length of the chordae and leaflets is smaller than the size of the annulus. This occurs with leaflet restriction due to rheumatic mitral regurgitation wherein the fibrosis has caused shortening of the chordae with a normal-sized annulus. Similar findings of a central jet occur as a consequence of coronary artery disease, cardiomyopathy, or any left ventricular dysfunction that causes the papillary muscles to be spatially displaced. In such cases the actual leaflet-chordal length may be normal but relative to the size of the annulus, and with the outward displacement of the papillary muscles the coapting leaflet edges are displaced apically into the ventricle from the annular plane. This has been decribed as "tenting" of the mitral valve. Rheumatic or ischemic mitral regurgitation may also cause a pattern of anterior leaflet "override" when the posterior leaflet is restricted more than the anterior leaflet, which also causes a posterior jet direction. Occasionally eccentric jets occur wherein the jet originates in the body of the leaflet rather than at the coaptation line. Theses are usually secondary to leaflet perforation. An eccentric jet direction may also be seen in patients with congenital mitral regurgitation related to a cleft mitral valve or in patients with

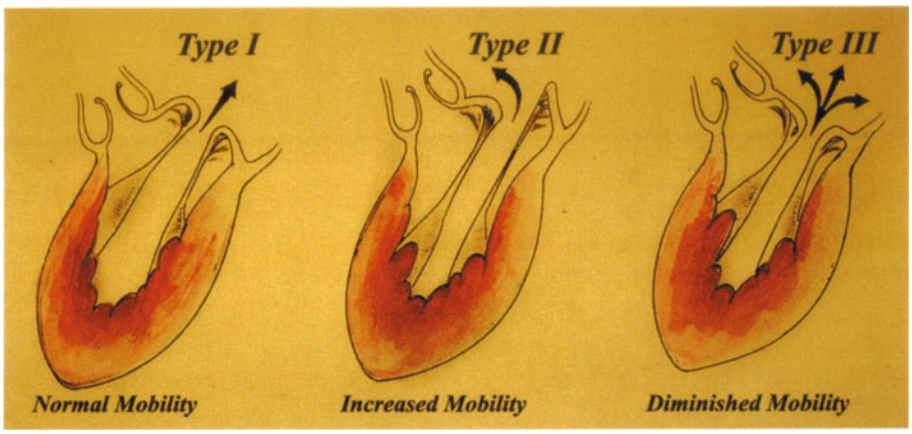
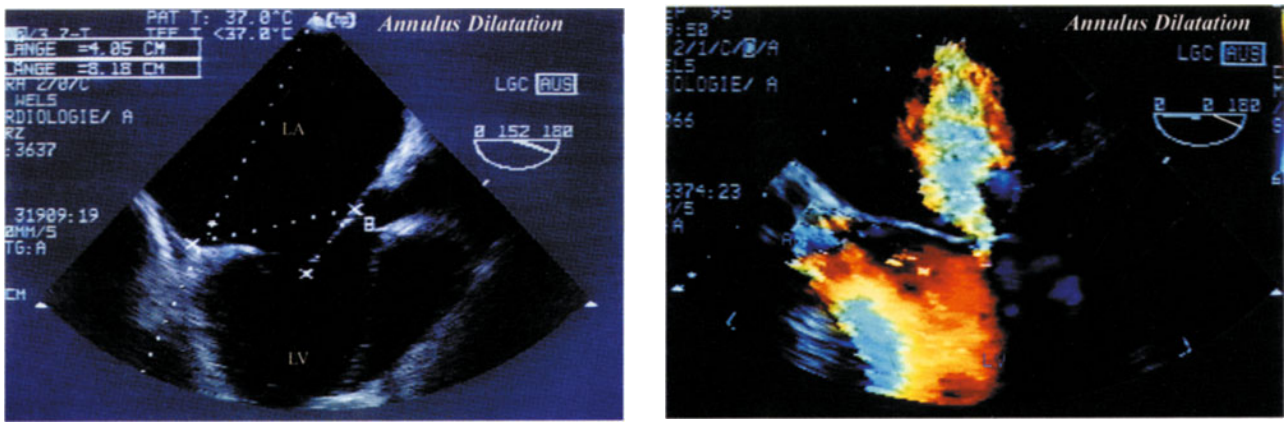


FIG. 1.1. Classification of leaflet mobility, Type I, II, III (TEE long-axis view)



a **b**

FIG. 1.2a. Type I. Mitral annulus dilatation is marked by a pathological ratio between the annulus length (here 8.15 cm) and the length of the anterior leaflet (4.05 cm) of >1.5, with normal leaflet mobility. This frequently results in mitral regurgitation (MR); **b** This coaptation gap causes a central regurgitation jet as demonstrated by color Doppler echocardiography

failed valve repair as a result of dehiscence of the leaflet suture line.

Careful scanning different image planes can sometimes visualize two or more jets within the left atrium. These patients may have flail or elongated chordae to both leaflets and these jets appear central or broad in direction.

Carpentier pragmatically classified valve dysfunction according to the range of motion of the leaflets, a system we currently use for echocardiographic classification [16]. The understanding of mitral valve function and pathophysiology brought by Carpentier, whose dysfunction-lesion-etiology concept has stimulated the development of a versatile arsenal of surgical techniques adapted to the multiple lesions responsible for mitral regurgitation.

Normal leaflet mobility, Type I (Fig. 1.2a,b)

This form of mitral insufficiency is caused by simple *dilatation of the annulus*, which reduces the coaptation surface of the leaflet to the extent that stable closure during the entire duration of

systole is not ensured. Usually, this form of mitral insufficiency is associated with a dilated ventricle. The coaptation gap causes a characteristic central regurgitation jet to occur that is well depicted on TEE-color Doppler, particularly in the long-axis view. On the echo image, the annulus diameter is compared with the anterior leaflet length (diameter/leaflet length = ratio). A ratio greater than 1.5 is interpreted as annulus dilatation.

Mitral insufficiency caused by excessive leaflet motion, Type II (Fig. 1.3a,b)

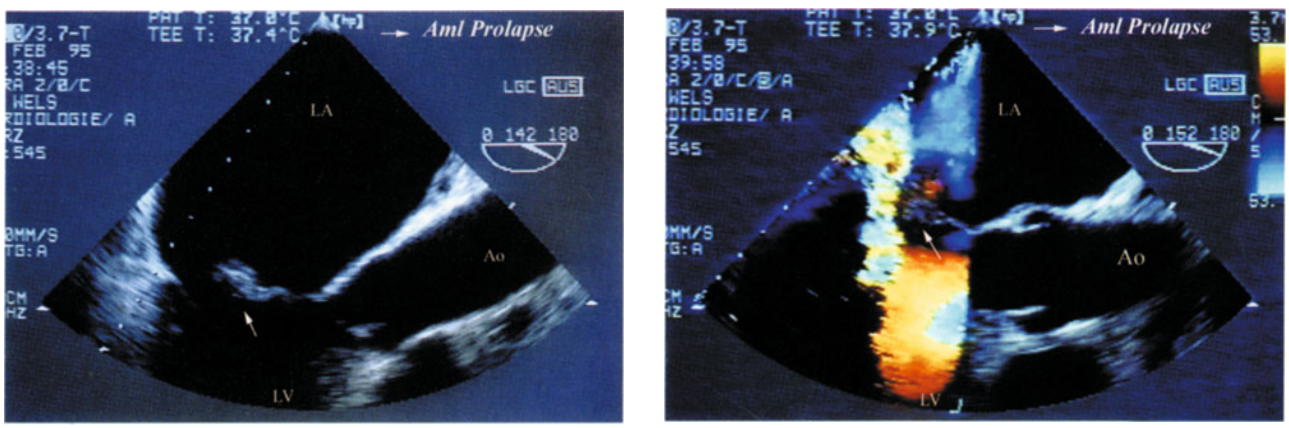
This is a frequent form of mitral insufficiency. It is caused by a leaflet prolapse and is frequently observed in association with chordal elongation or chordal rupture, or in association with rupture of a papillary muscle. Either of the two leaflets may be prolapsed. The regurgitation jet on color Doppler is seen along the surface of the non-prolapsed leaflet. However, both leaflets may also prolapse simultaneously (bi-leaflet prolapse or multiple prolapse/billowing as in Barlow's disease).

Mitral insufficiency in restricted leaflet motion, Type III (Fig. 1.4a,b)

This occurs when the leaflet is shrunken or restricted in its mobility, so that its insufficient flexibility does not permit rapid closure of the valve at the end of diastole or, that the available leaflet surface has become too small to permit complete closure of the valve. This pathology is encountered in rheumatic valve pathology and in ischemic heart disease. If the posterior leaflet is affected (which is the usual case), color Doppler will reveal severe mitral regurgitation along the surface of the posterior leaflet, which spreads further in central direction as the coaptation gap increases, and may fill the entire atrium.

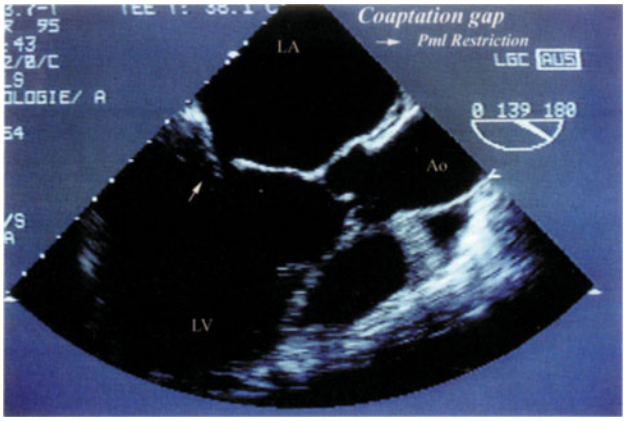
Nomenclature of mitral leaflet: "billowing," "prolapse," "floppy," and "flail"

Appropriate patients selection for successful surgical repair of the mitral valve depends on the special location and mechanism of regurgitation,



a **b**

FIG. 1.3a. Type II. Leaflet prolapse, here of the anterior leaflet, secondary to chordal elongation; **b** Regurgitation jet on color Doppler along the surface of the posterior leaflet



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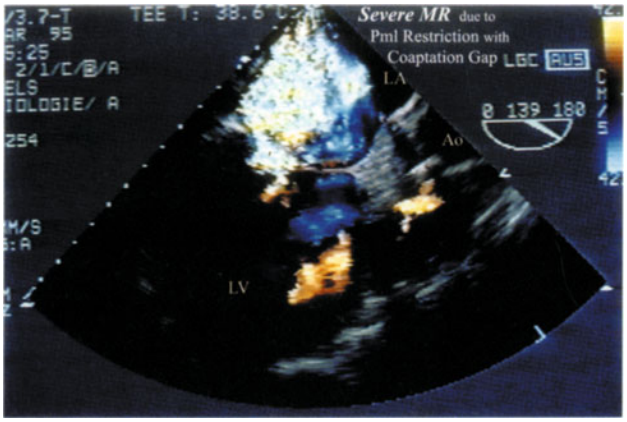


b

FIG. 1.4a. Type III. Leaflet restriction frequently affects the posterior leaflet (arrow) after fibrotic shrinkage in rheumatic disease or is due to ischemic papillary muscle dysfunction after posterior wall infarction; **b** Color Doppler shows severe mitral regurgitation alongside the surface of the posterior leaflet. As the coaptation gap increases, the regurgitation spreads in central direction and fills the entire atrium

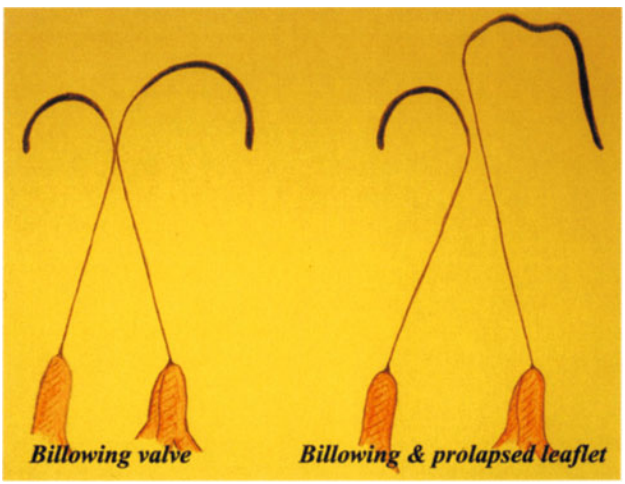


FIG. 2.1. Billowing valve

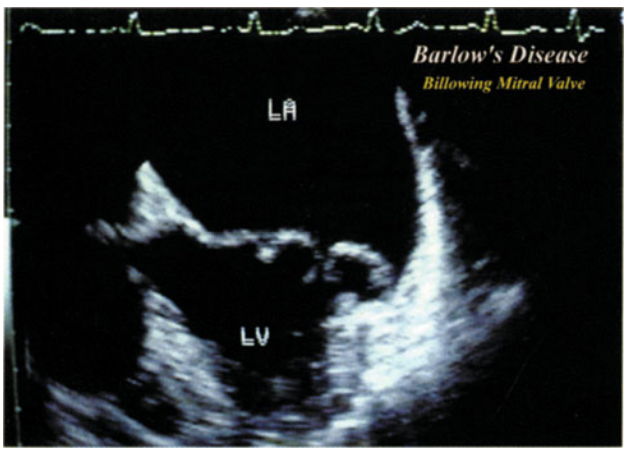


FIG. 2.2. A typical billowing mitral valve with myxomatous altered tissue

which, in turn, has necessitated us to define for the terms "billowing," "prolapse," "floppy," and "flail" leaflet (Fig. 2.1–6).

The normal mitral leaflets billow slightly, after closure, into the left atrial cavity. Exaggeration of this, which may range from being marked and diffuse to mild and involving only one scallop or even part of a scallop, should be termed a "*billowing mitral leaflet*." If this billowing mitral leaflet progresses, failure of leaflet edge apposition may supervene and then there is "*mitral valve prolapse*." With the mitral valve prolapse, there has to be some mitral regurgitation, which is reflected clinically by a mitral systolic murmur and confirmed on echocardiography. If the billowing mitral leaflet is extreme with very voluminous leaflets and elongated chordae, the term "*floppy*" is appro-

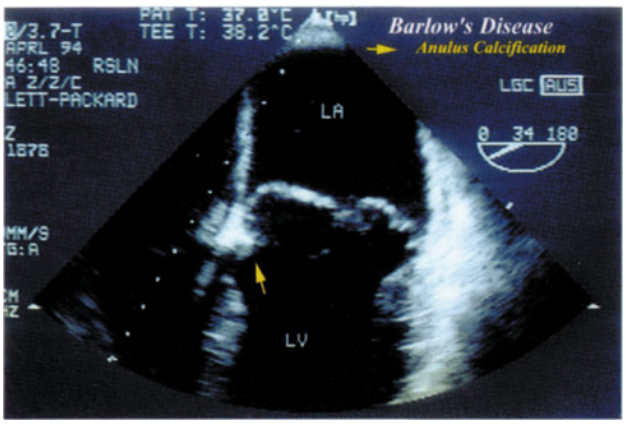


FIG. 2.3. Barlow's disease with a billowing valve and leaflet prolapse, as seen in Fig. 2.1 on the left. The operation is frequently complicated by the presence of annulus calcification (as marked here by an arrow in 4 chamber view-TEE)

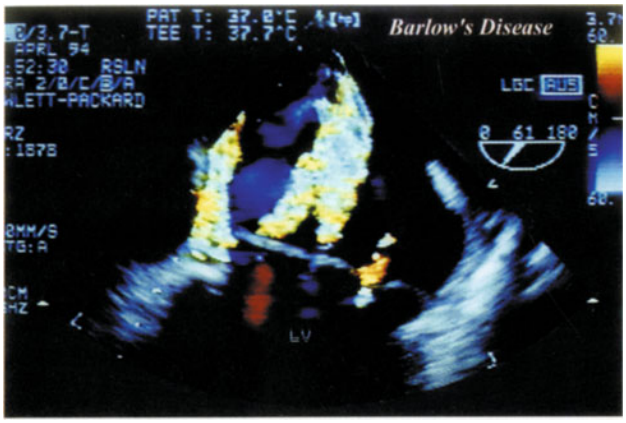


FIG. 2.4. Multiple regurgitation jets on color Doppler due to differing mobility of billowing and leaflet prolapse, typical characteristics of Barlow's disease

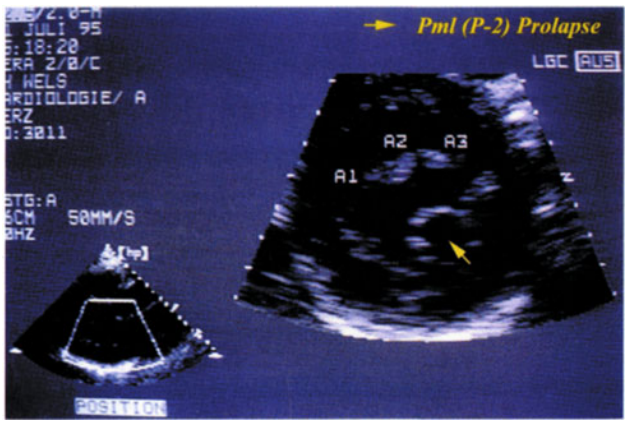


FIG. 2.5. Arrow directs towards a prolapse of the middle scallop (P2) of the posterior leaflet. Short-axis view on the TTE cross-section showing different leaflet scallops for segmental leaflet analysis

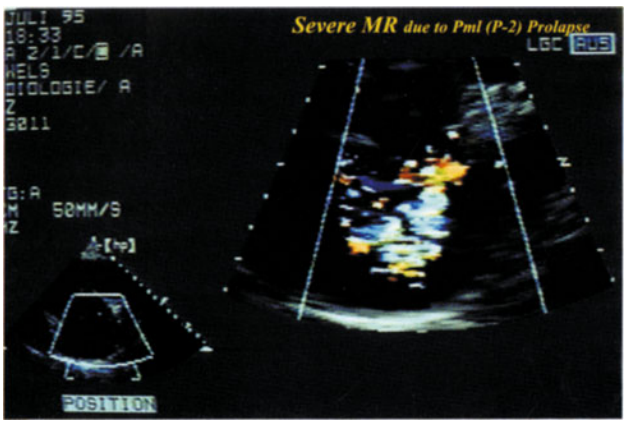


FIG. 2.6. Regurgitation on color Doppler: The backward flow is exclusively limited to the prolapsed P2 segment

priate. Some billowing mitral leaflet is almost always present with floppy valve. Echocardiographic confirmation of mitral valve prolapse entails essentially the demonstration that the leaflets edges are disengaged. Progression of the mitral valve prolapse with a floppy valve or with a billowing mitral leaflet may result in ruptured or at least grossly elongated chordae, and part of the leaflet will then be "*flail.*" Mitral regurgitation is now hemodynamically significant, and the displacement of the leaflet edge is readily apparent on echocardiography. "Floppy" connotes the extreme of marked billowing mitral leaflet and "flail" that of severe mitral valve prolapse.

A **"billowing and prolapsed"** mitral valve is a typical complication of the condition described by Barlow and Carpentier, known as Barlow's disease. It is the result of abnormal enlargement of the leaflet, elongation of the chordae and dilatation of the valve annulus (Fig. 2.1–6). In terms of pathological anatomy, one finds a "myxoide degeneration". These features can easily detected by TEE or TTE using different

imaging planes. The method serves to detect individual segment movements and fragmental or prolapsed commissures. With the help of color Doppler, regurgitant jets in any pathological section can be localized. The short-axis view on the TTE cross-section plays a key role in preoperative preparation [15].

Mitral regurgitation

The complex valve mechanism may become incompetent from several causes that produce pathological or functional changes in one or more components of the valve. The leaflets may be fibrosed and shortened as a result of rheumatic carditis. Alternatively, leaflet tissue may be excessive owing to myxomatous degeneration and thus fail to maintain coaptation. Myxomatous degeneration may also produce dilatation and impaired contraction of the annulus, which is a feature of the floppy valve and Marfan's syndromes. Marked dilatation of the annulus occurs in severe rheumatic carditis with resultant significant mitral regurgitation. Failure of the normal contraction of the annulus probably contributes to mitral regurgitation in early active rheumatice carditis and in primary mitral valve prolapse. Mitral regurgitation may sometimes be associated with calcification of the annulus. Elongated or ruptured chordae tendineae will allow one or more leaflets to become flail and may result from rheumatic carditis, infective endocarditis, myxomatous degeneration, or idiopathic causes. Lastly, the papillary muscles may not contract normally and thus fail to maintain coaptation of the leaflet edges. Papillary muscle dysfunction may be due to other causes but is most commonly a consequence of occlusive coronary artery disease, which may also cause these muscles to rupture.

3 Principles of reconstruction

Mitral valvular heart disease most often is a chronic illness. In these cases, patients are treated conservatively until the time when symptoms or incipient pulmonary hypertension or ventricular dysfunction indicate the need for surgical intervention. However, on some occasions, mitral valvular heart disease presents itself as an acute illness due to sudden changes in intrinsic valvular function. In these cases, therapeutic decisions must be accomplished rapidly, and the underling pathophysiological disorders always require prompt assessment.

Symptoms are not the only indication for surgery in patients with mitral valve disease. Other variables include severity of valvular dysfunction, left ventricular function, atrial fibrillation, pulmonary hypertension, history of or risk for thromboemboli, age, cause of dysfunction, pathoanatomy, overall patient health, and other cardiac conditions. Many patients have severe mitral regurgitation before recognizable symptoms, and substantial left ventricular dysfunction may occur in asymptomatic patients. Waiting for progression to functional class III often subjects the patient to prolonged left ventricular volume overload, resulting in permanent myocardial dysfunction, which contributes to increased operative mortality and poor clinical outcome. The onset of significant congestive symptoms warrants consideration for surgery, even if the symptoms are mild.

Early surgery may remove the volume overload and prevent progressive myocardial dysfunction, pulmonary hypertension, left atrial enlargement, arrhythmias, interval complications, or sudden death. It has the additional advantage of intervening when the valve disease is less advanced and more amenable to repair. In addition, a younger patient is better able to tolerate surgery. The continuing decline in operative mortality with cardiac surgery and the increased degree of success with mitral reconstruction are compelling arguments for early surgical intervention before irreversible complications of mitral valve disease develop.

The concept of comprehensive valvuloplasty is based is that diseased component of the mitral valve apparatus: the annulus, leaflets, papillary muscles, and chordae tendineae. Correction of only one abnormal component is an incomplete repair. Therefore, surgery to restore valvular function should be directed at repair of all affected components of the mitral valve.

The goal of reconstruction in all forms of mitral insufficiency is to achieve a widest possible surface of coaptation along the entire line of coaptation with no localized leaflet prolapse at any site. This is the final goal of any repair method of surgery. A wide coaptation of the leaflets in the early ventricular systole absorbs a major portion of the energy discharged by the ventricle on the undersurface of the leaflet during systole. *After successful repair, the curved coapting closure line can be compared with the mouth of a "smiling valve"* [16].

4 MITRAL VALVE SURGERY IN ANNULUS CALCIFICATION (Figs. 3–8)

Marked calcification of the mitral annulus impairs its motion during the cardiac cycle. Dense calcification of the entire submitral region, in its most severe form, is characterized by the development of a rigid curved ring of calcium that encircles the mitral orifice. Ramifications of these calcifications, so-called calcific spurs, often project into the adjacent left ventricular myocardium (Fig. 3.1–8). The calcification may also affect the basal portions of the valve leaflet and cause distortion of the posterior mitral valve leaflet with limitation of motion. The base of the posterior mitral valve leaflet is pushed towards the atria, often stimulating a degree of mitral prolapse. This may result in insufficient leaflet area for coaptation, eventually leading to valve insufficiency which, if untreated, may result in cardiac failure. Severe mitral insufficiency in the presence of annulus calcification is usually caused by ruptured chordae tendinae or by the presence of elongated chordae tendinae that partly prolapse into the atria (Fig. 4.1–2). Mild or moderate degrees of mitral regurgitation are commonly a functional consequence of submitral calcification. Occasionally, such calcification lead to functional mitral stenosis with a significant transvalvular gradient during diastole. Anatomic distortion of valve may predispose to endothelial lesions, necrosis, infection and thrombus or vegetation formation with an increased risk for systemic emboli (Fig. 4.3). There is a linear relationship between the occurrence of cerebrovascular accident and the severity or extent of mitral annulus calcification. One of the most common causes of cerebrovascular accident secondary to thromboembolism is atrial fibrillation, which is much more common in patients with coexisting annulus calcification. Other known complications of extensive mitral annulus calcification include the development of bacterial endocarditis as the calcific masses erode and ulcerate onto the endocardial surface. Furthermore, atrioventricular conduction abnormalities caused by extension of the calcification process into the ventricular septum may occur. Calcification secondary to mechanical trauma, encroaching on the bundle of His, and the consecutive occurrence of symptomatic AV blocks and intraventricular conduction abnormalities is known as Lev's disease [17], [18].

4.1 Endoventricular pericardial patch annuloplasty (Figs. 4–6)

Once the basic pathological change was identified, decalcification is performed as an *en-bloc resection* to prevent calcium fragmentation. The surgical procedure is started by performing an incision in the atrial endothelium along the margins up to the calcific spurs, using a sharp knife [19]–[22]. Extreme care is taken during each step to avoid disrupting the atrioventricular continuity, rupture of the ventricular wall during excision of the calcified papillary muscle tissue, or damage to the circumflex artery (Fig. 5.1–4). After removal of the mobilized calcified tissue, the area is amply irrigated to minimize the risk of embolization. Subsequently, a quadrangular resection of the posterior mitral valve leaflet with complete detachment from commissure to commissure is carried out. After "en-bloc decalcification" of the mitral annulus and the adjacent myocardium, fragile or fibrous tissue is exposed at the superior atrial and the inferior ventricular end. Further reconstruction of the mitral annulus is performed by means of extended *endoventricular pericardial annuloplasty*, for which an autologous pericardial patch was used (Fig. 4.4, Fig. 6.1–4).

4.2 Autologous pericardial patch

The autologous pericardial patch usually a rectangular strip measuring 4 × 6 cm – is harvested from the pericardium above the right atrium and

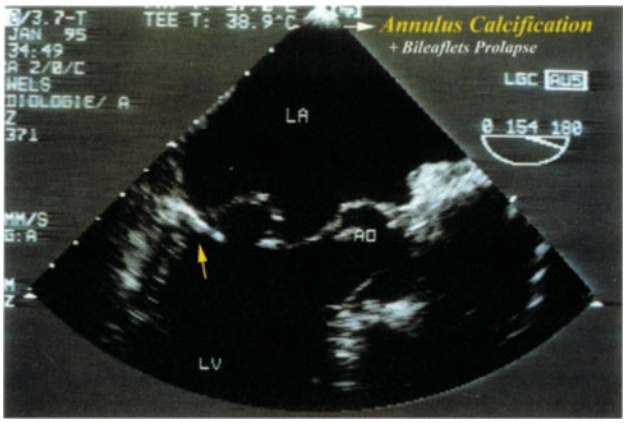


FIG. 3.1. Multiplane TEE long-axis view shows prolapse of both leaflets with a calcified annulus and calcific spurs projecting into the adjacent myocardium of the left ventricle

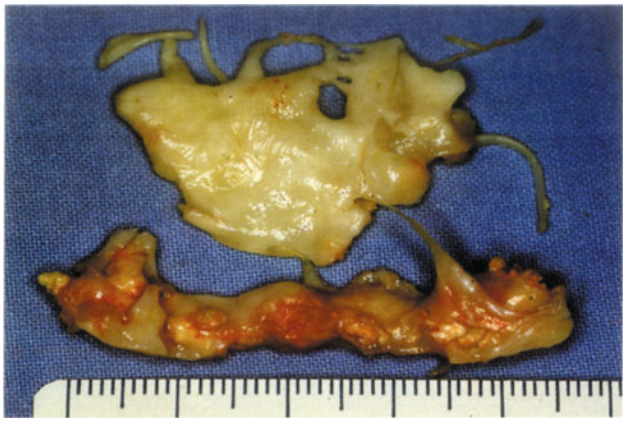


FIG. 3.2. Calcified annulus, 4.9 cm in width, with a perforated leaflet after en-bloc resection

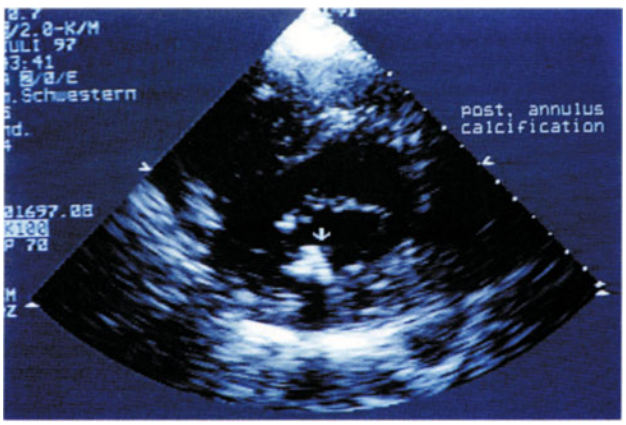


FIG. 3.3. Cross-section TTE short-axis view shows (arrow) a severely calcified posterior leaflet and annulus

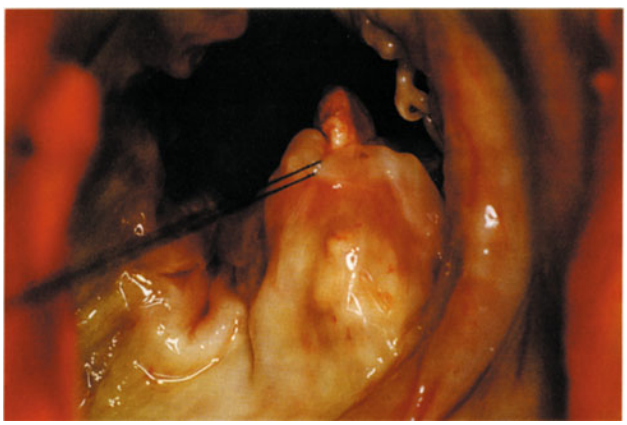


FIG. 3.4. Intraoperative image viewed from the left atrium shows the calcification process

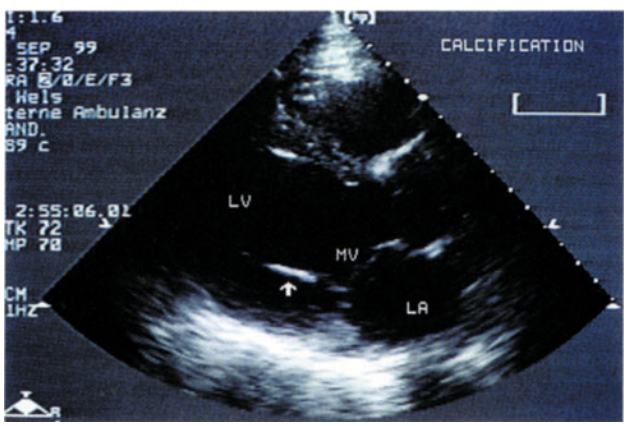


FIG. 3.5. Transthoracic long-axis image indicates a calcified tendinous bundle and papillary muscle (arrow)

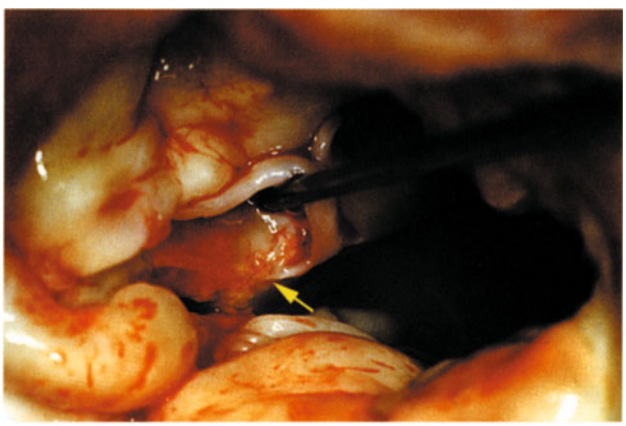


FIG. 3.6. Intraoperative photo of the subvalvular apparatus, as visualized by help of nerve hooks, demonstrates a calcified papillary muscle

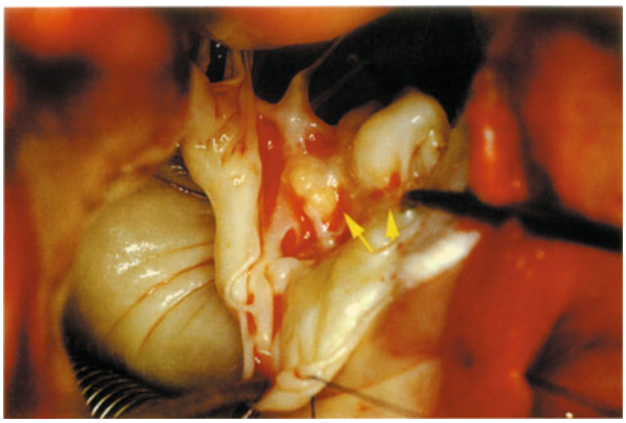


FIG. 3.7. Surgical photograph depicts a calcified leaflet and annulus, as viewed from the ventricular aspect

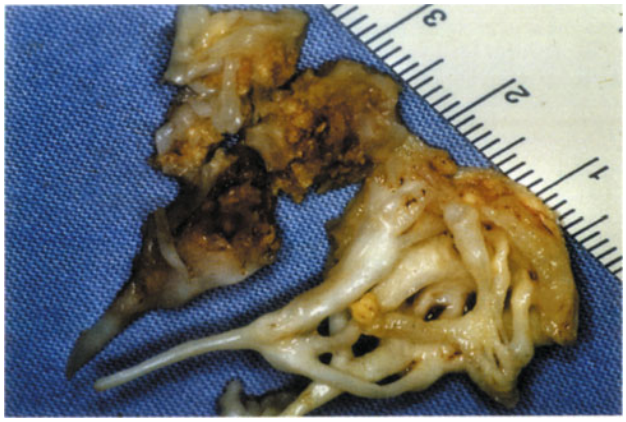


FIG. 3.8. Calcified bundle of chordae tendineae and papillary muscle after resection in the same patient

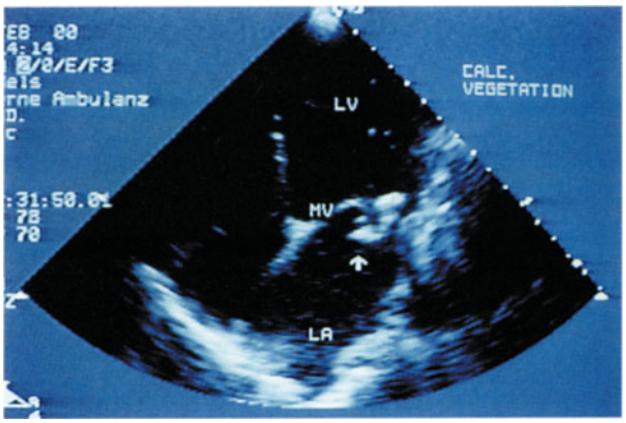


FIG. 4.1. The arrow in the apical 4-chamber view transthoracic echocardiography (TTE) shows calcified vegetations of the posterior leaflet with concomitant chordal rupture and calcified annulus

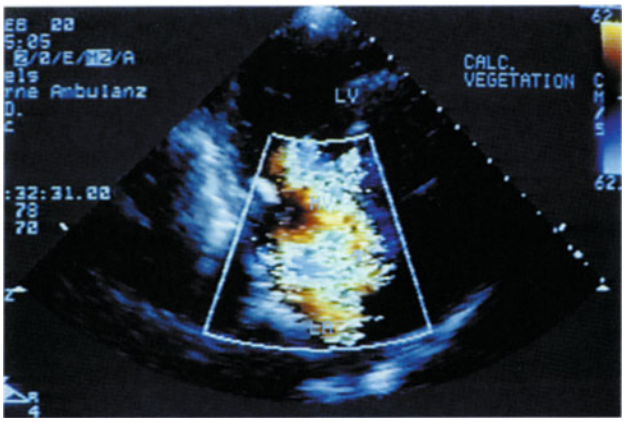


FIG. 4.2. Severe mitral valve regurgitation as visualized by color Doppler in the long-axis view

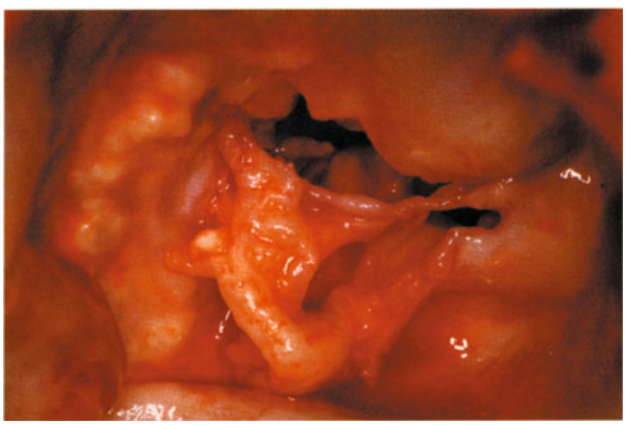


FIG. 4.3. The surgical pathology confirms the echocardiographic findings. The two arrows on the left show commissural calcification

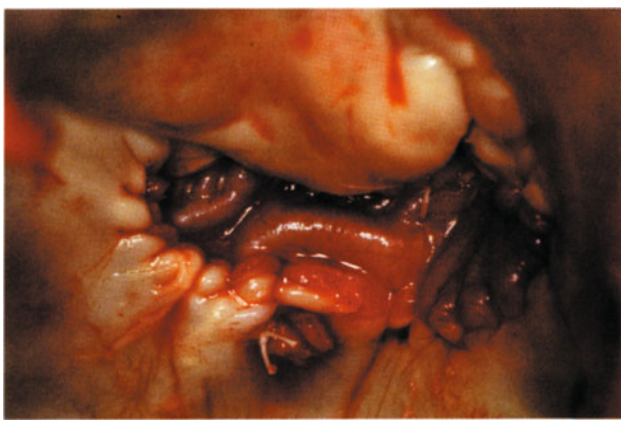


FIG. 4.4. Picture demonstrating successful reconstruction of the annulus and leaflet with endoventricular pericardial patch annuloplasty after en-bloc resection

Possible surgical complications of calcified mitral annulus

FIG. 5.1. The illustration demonstrates surgical risk of atrioventricular rupture and damage to the circumflex coronary artery

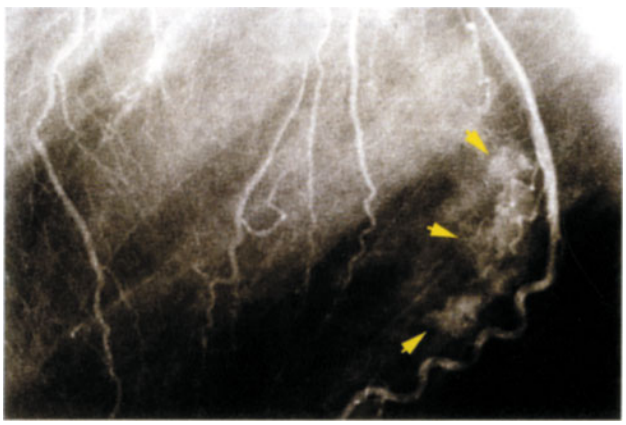
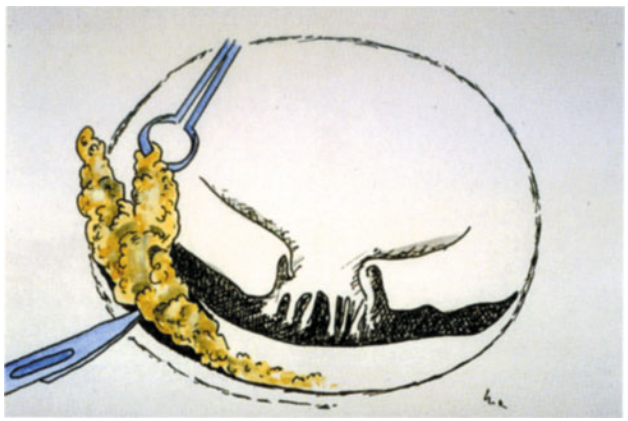
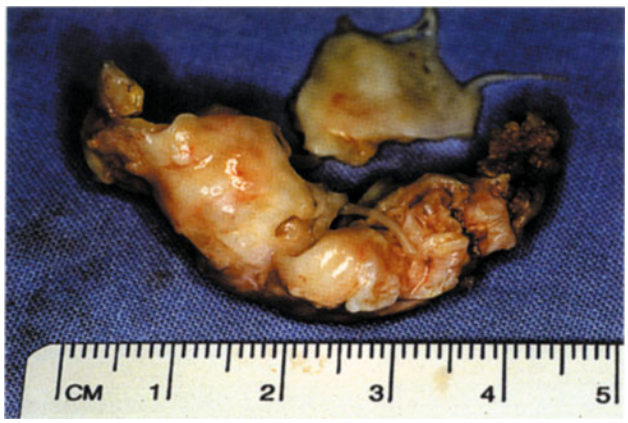


FIG. 5.2. Routine angiography demonstrates close proximity of left circumflex artery and horseshoe calcification



5.3



5.4

FIGS. 5.3 and 5.4. Decalcification by means of sharp en-bloc resection. This permits more exact dissection to incise the endocardium along the calcified horseshoe and also prevents calcium fragmentation

Endoventricular

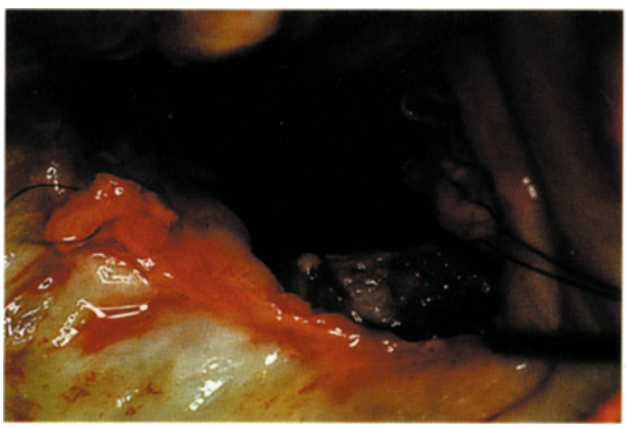


FIG. 6.1. After complete removal of the calcified mass, an endoventricular endocardial annuloplasty with a pericardial patch is performed

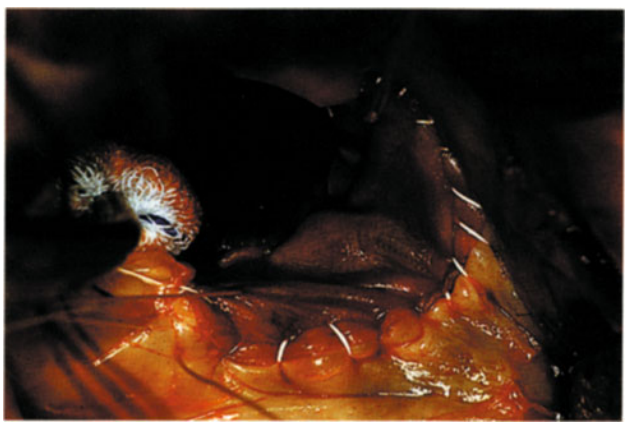


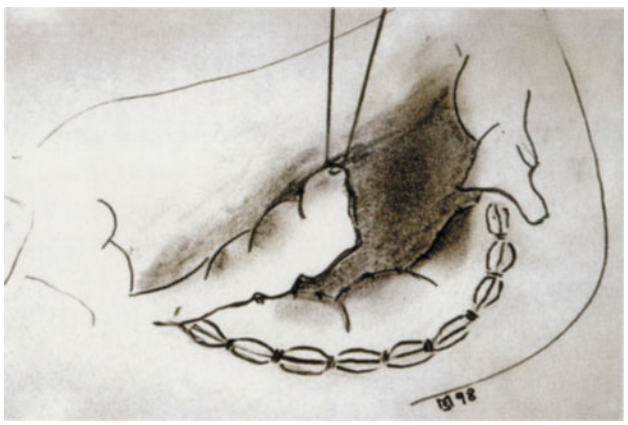
FIG. 6.2. The patch is sutured to the fibrotic endocardium of the atrium and to the left-ventricular outflow tract

is cleaned of pleural tissues. The patch is fixed in a 0.62% glutaraldehyde-buffered solution for 30 minutes and then rinsed with saline solution in three separate bowls for an additional 30 minutes. The smooth surface of the pericardium is turned toward the atrium for valve repair. The surgical technique of patch application varies depending on the site of the lesion [23].

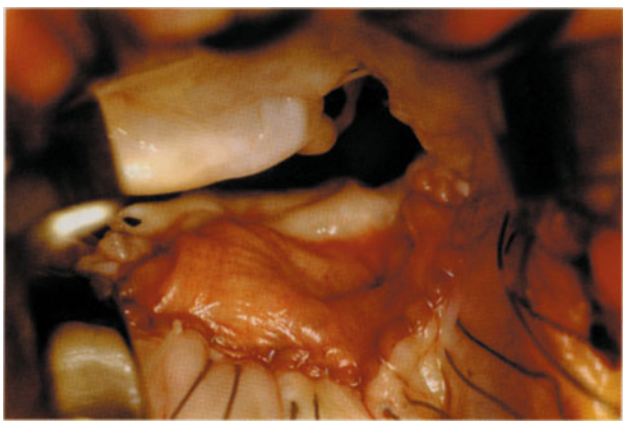
A suitable strip of autologous pericardium is tailored and its margins sutured to the fibrotic endocardium of the left ventricular and posterior left atrial wall with continuous 5-0 or 6-0 polytetrafluoroethylene (PTFE) sutures. Defects created by the quadrangular leaflet resection are repaired by means of plastic leaflet closure and by leaflet advancement to the appropriate level, using 6-0 or 7-0 PTFE sutures (Fig. 6.2–4).

4.3 Sliding atrioplasty (Fig. 7.1–4)

Sliding atrioplasty is an alternative method of mitral valve reconstruction for a calcified poste-



6.3



6.4

FIGS. 6.3 and 6.4. Show a surgical illustration of valve repair with the re-sutured leaflet and a pericardial patch to prevent atrioventricular rupture

Sliding atrioplasty

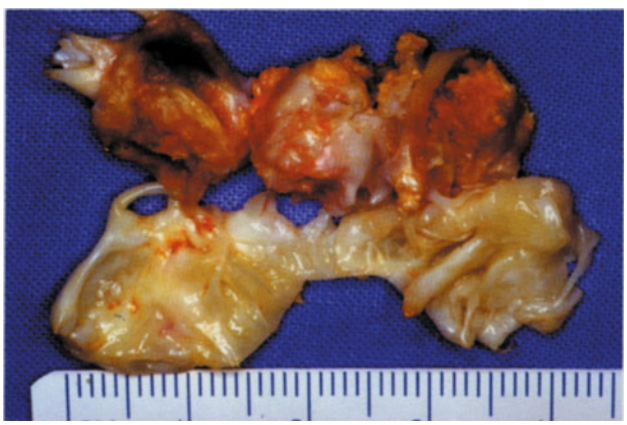


FIG. 7.1. Removal of more than 4 cm of the calcified annulus and the corresponding posterior leaflet

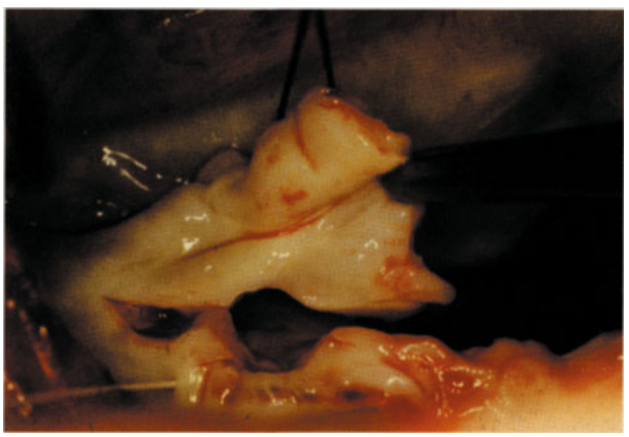


FIG. 7.2. After decalcification of the atrium and annulus, the edge of the atrium is mobilized to form a flap at the atrioventricular junction

rior leaflet and annulus without serious calcification of the underlying myocardium [19], [22]. After decalcification of the atrium, an atrial edge is mobilized to form a flap at the atrioventricular junction. To construct a new mitral annulus, this flap is then employed to cover the underlying myocardium, using gently tied multiple figure-of-eight 6-0 or 7-0 polytetrafluoroethylene vertical sutures. In accordance with the "sliding leaflet" technique, the leaflet remnants are sutured to the reconstructed mitral annulus (Fig. 7.2–4).

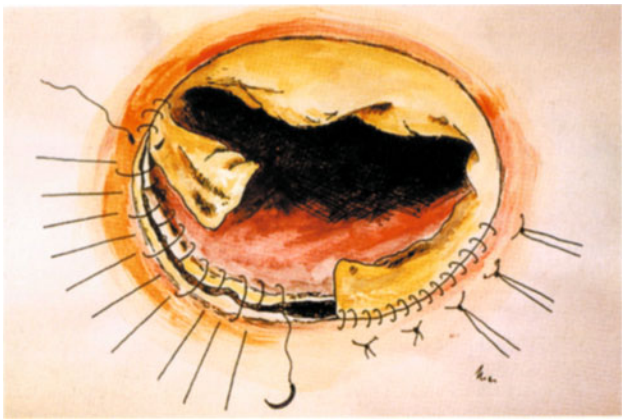


FIG. 7.3. Surgical illustration shows two layers of fibrotic tissue, reconstructed with figure-of-eight vertical sutures, followed by closure with posterior leaflet tissue

4.4 Rotation paracommissural sliding plasty (Fig. 8.1–6)

Rotation paracommissural sliding plasty of the valve leaflet is indicated in patients with calcified leaflet commissures (Fig. 8.1). After resection of the mineralized or infected valve area, both anterior and posterior leaflets are detached from the annulus (Fig. 8.2–4), allowing them to slide to fill the resection defect and to restore function. Usually the posterior mitral valve leaflet is further mobilized, being incised along its annular attachment. A new commissure is created by appropriate clockwise or counterclockwise rotation of the mobilized posterior leaflet, using an interscallop indentation as the new commissure (Fig. 8.5). The indentation between scallops is well suited for this purpose because leaflet distortion is avoided due to preservation of the functional chordal architecture [24]. A great reduction of the annulus diameter is avoided in these patients and yet sustained successful elimination of the severe mitral insufficiency is achieved (Fig. 8.6).

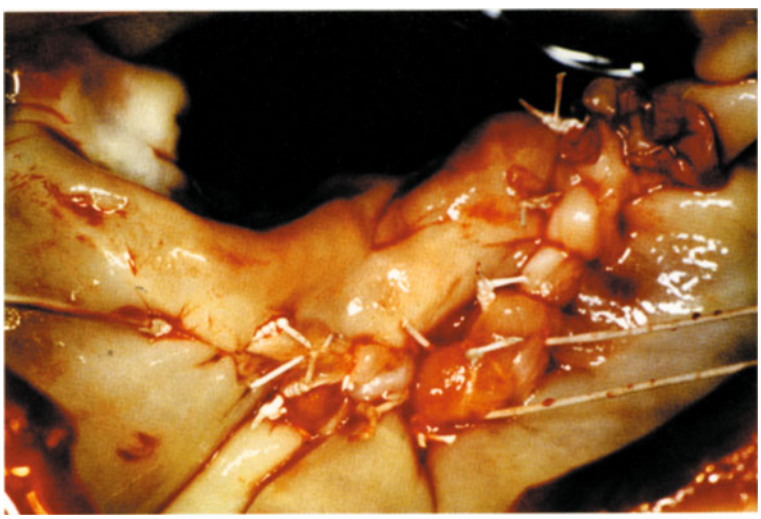
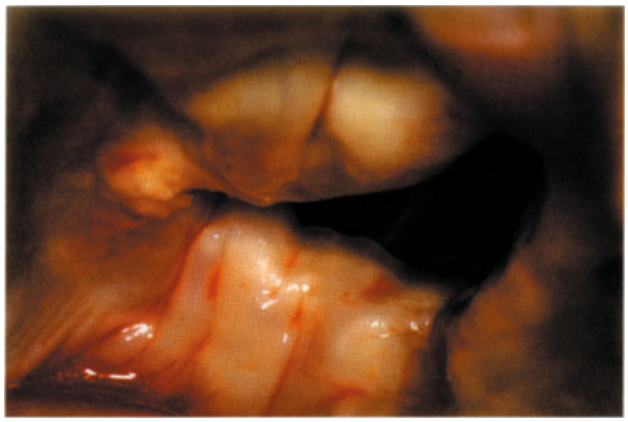
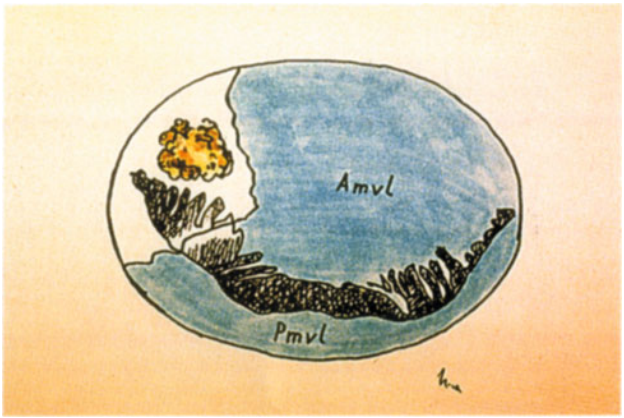


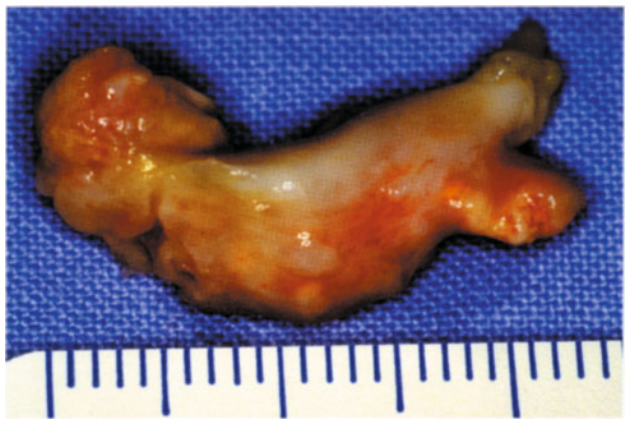
FIG. 7.4. The sliding technique is conducted by suturing the valve leaflet remnants to the reconstructed mitral annulus



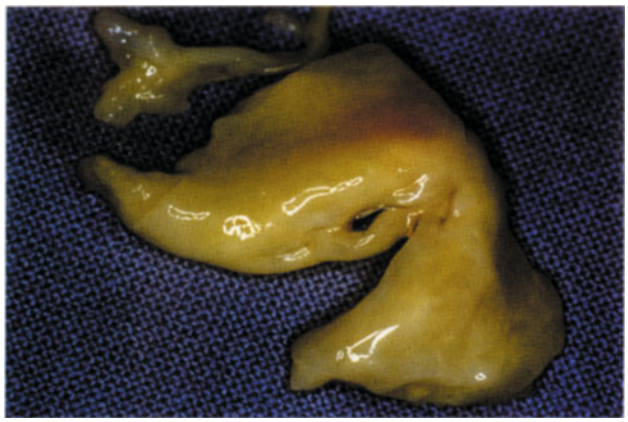
8.1



8.2



8.3



8.4

FIG. 8.1. Calcified commissure secondary to rheumatic disease, with severe mitral insufficiency

FIGS. 8.2–4. Calcified segment and pathological area due to prolapse and infectious tissue from the anterolateral and posteromedial commissure was extensively resected

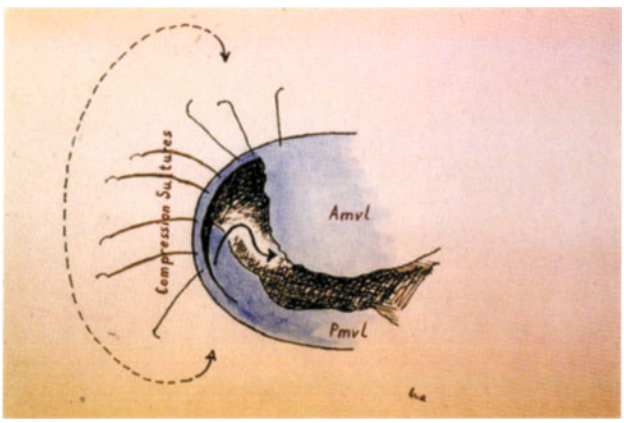


FIG. 8.5. Technique of rotation paracommissural sliding plasty. Extensive detachment of the posterior leaflet up to the middle scallop is performed; the arrows demonstrate the convergence of the two leaflets after mobilization

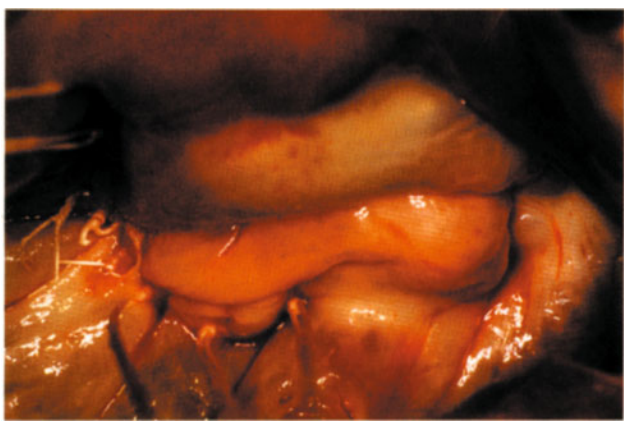


FIG. 8.6. Leaflet coaptation after rotation plasty

Mitral valve surgery in annulus calcification

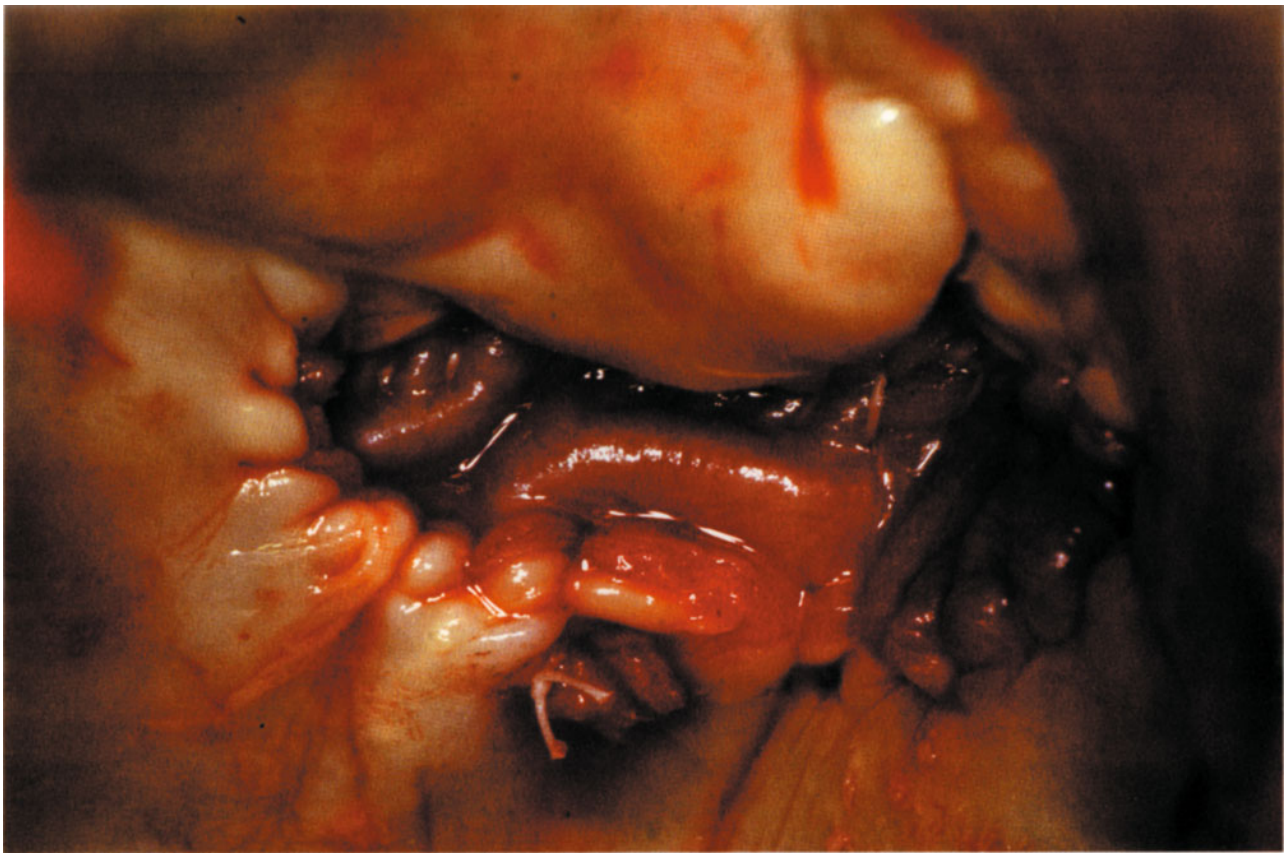


FIG. 4.4 (enlargement). Picture demonstrating successful reconstruction of the annulus and leaflet with endoventricular pericardial patch annuloplasty after en-bloc resection

Sliding atrioplasty

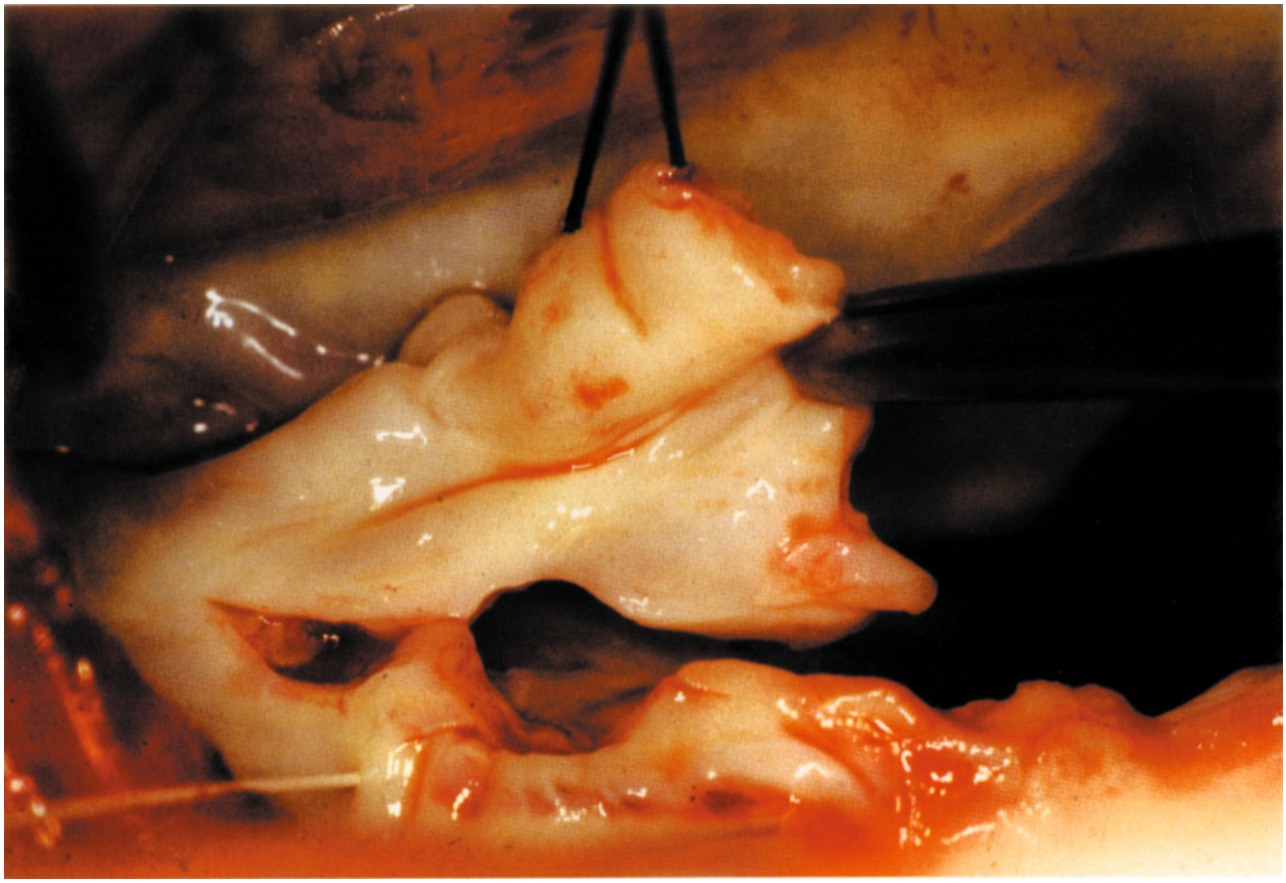


FIG. 7.2 (enlargement). After decalcification of the atrium and annulus, the edge of the atrium is mobilized to form a flap at the atrioventricular junction

Mitral valve surgery in annulus calcification

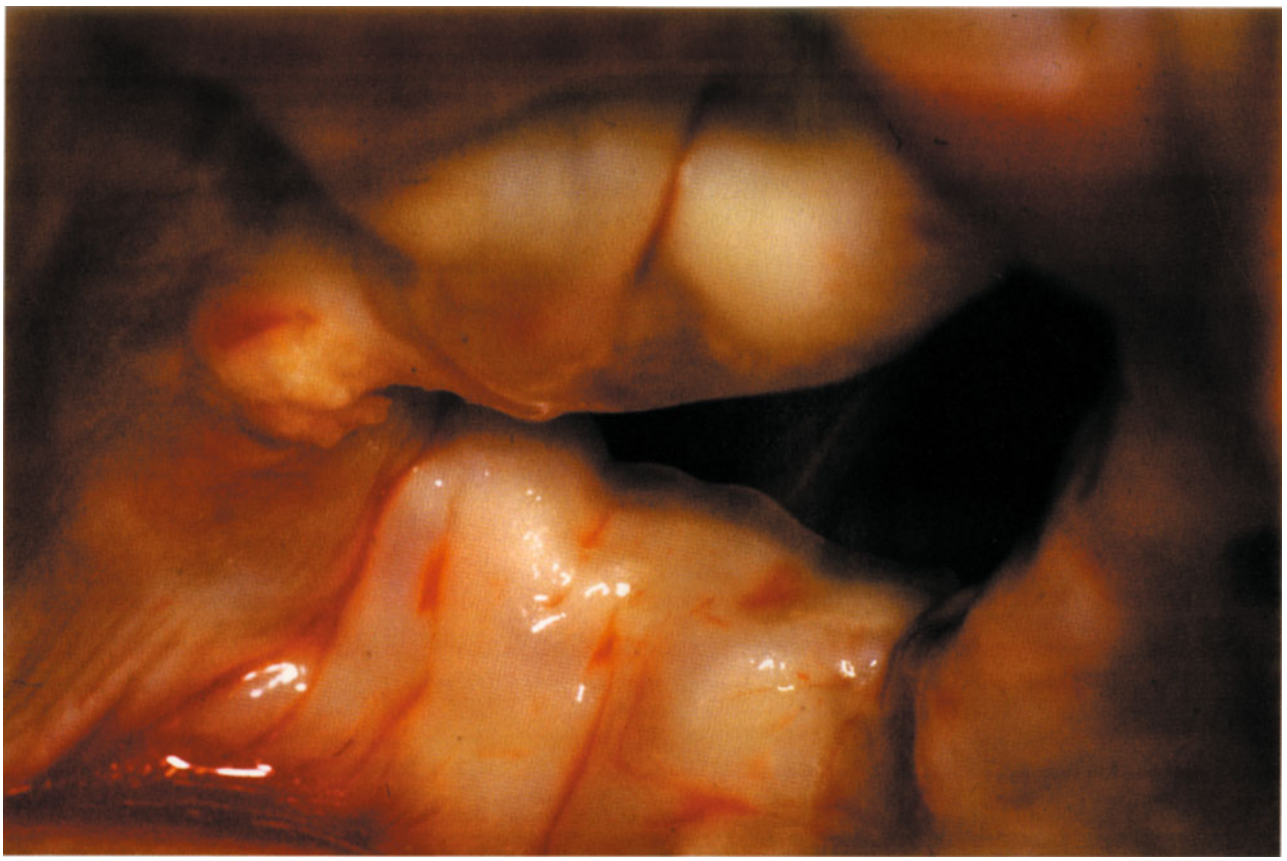


FIG. 8.1 (enlargement). Calcified commissure secondary to rheumatic disease, with severe mitral insufficiency

Rotation paracommissural sliding plasty

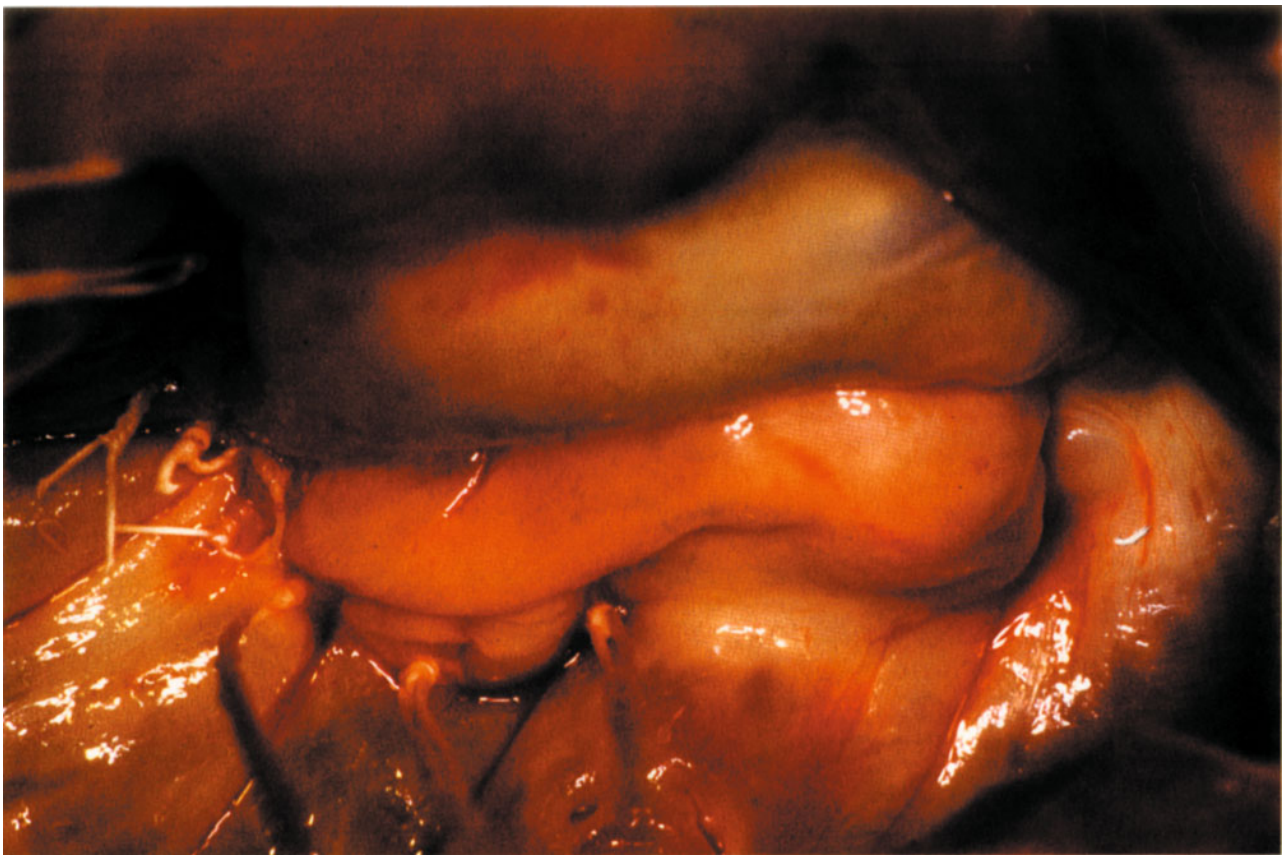


FIG. 8.6 (enlargement). Leaflet coaptation after rotation plasty

5 Mitral valve surgery in Barlow's disease

Barlow's disease is a primary or idiopathic degenerative condition of the mitral valve leaflet prolapse with excessive myxomatous altered tissue can be detected on histology. The reason for the onset of this degenerative condition is ill understood. It may commence in childhood, and young adults. The condition has a familial incidence in some cases and in others pectus excavatum or other abnormalities of the bony thorax suggest a generalized disorder of connective tissue. On the other hand, myxomatous degeneration is common in the elderly. It leads to the development of redundant valve tissue and is often associated with excessive elongation of the chordae tendineae or rupture of the chordae. The process may be focal, involving only one scallop or even a portion of one scallop, or it may be diffuse, involving both leaflets. The increase in transverse dimension results in the circumference of the leaflets being larger than the circumference of the mitral valve annulus (Fig. 9.1–4). In this context, severe *calcification* of the mitral annulus has been reported to occur with advancing age [25]. Likewise, it is known that systolic anterior motion of the mitral valve leaflet **(SAM)** after valve repair is a common occurrence in this disease, if the residual length of the anterior and/or posterior leaflet after repair is still too large. Especially after annuloplasty with implantation of a small composite annuloplasty ring, the left ventricular outflow tract is blocked because of annulus compression with simultaneous projection of leaflet tissue into the outflow tract (Fig. 10.1).

Currently, an increasing numbers of leaflet sliding repair are also performed, this type of repair having been introduced by Carpentier in order to prevent the problem of SAM, which occurred in 5–10% of patients with degenerative disease [16], [19], [20], [22].

5.1 Leaflet sliding plasty (Fig. 9.1–8)

When the posterior leaflet is > 1.5 cm long in patients having large and floppy leaflets, a sliding repair to avoid **SAM** is routinely applied. When using this technique, the quadrangular resection of all prolapsed scallops is completed first (Fig. 9.5). Following tissue resection, the leaflet is detached from the annulus until close to the commissure (Fig. 9.6). After performing several annulus compression sutures, the valve leaflet remnants can be connected to the annulus without tension and easily made to form a new leaflet. Provided the necessary surgical discipline

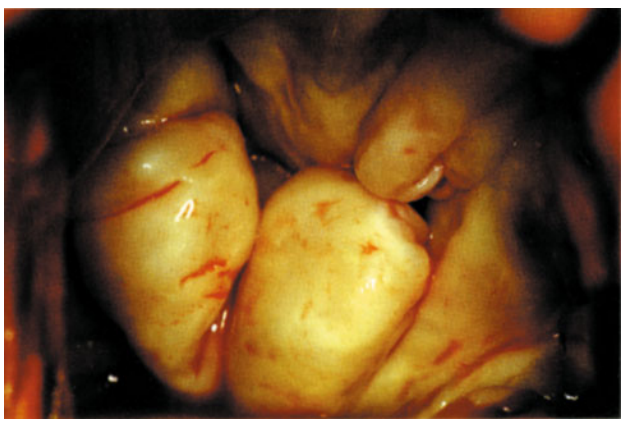


Fig. 9.1. Multiple excessive myxomatous tissue with annulus dilatation

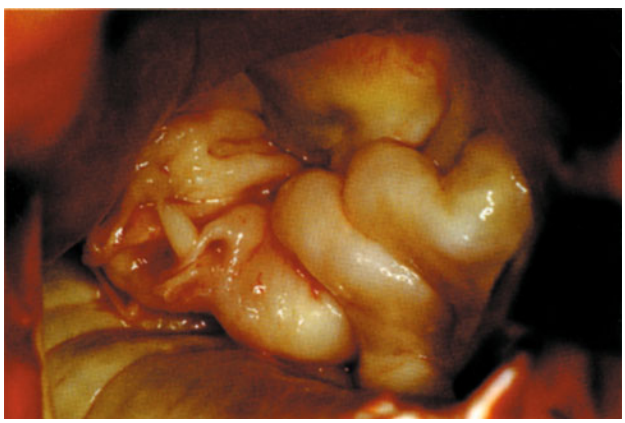


Fig. 9.2. A typical characteristic is a vast area of prolapsed tissue

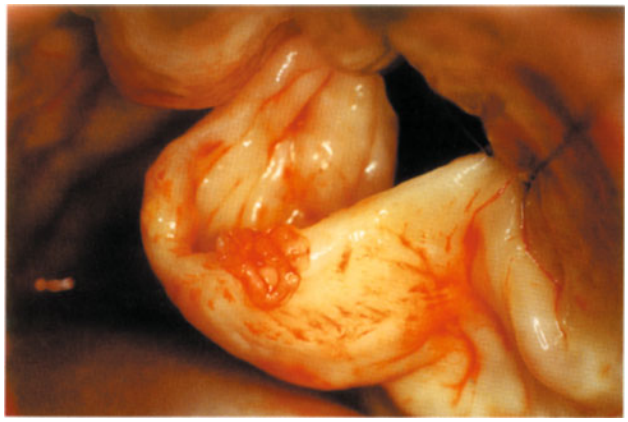


FIG. 9.3. Segmental chordal rupture is a frequent cause of severe mitral regurgitation after early degeneration

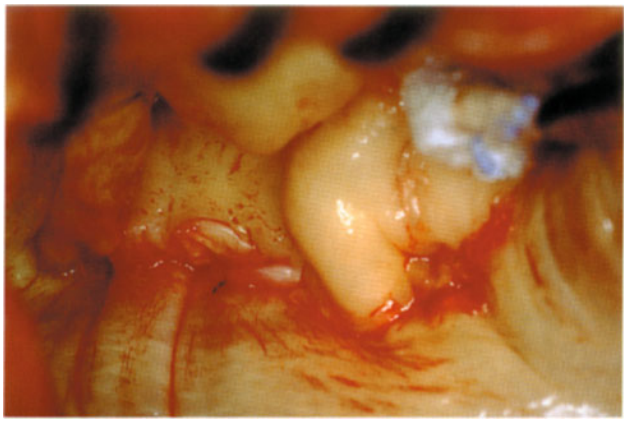


FIG. 9.4. Annulus calcification is a common feature (even at a young age or after recurrent annulus inflammation)

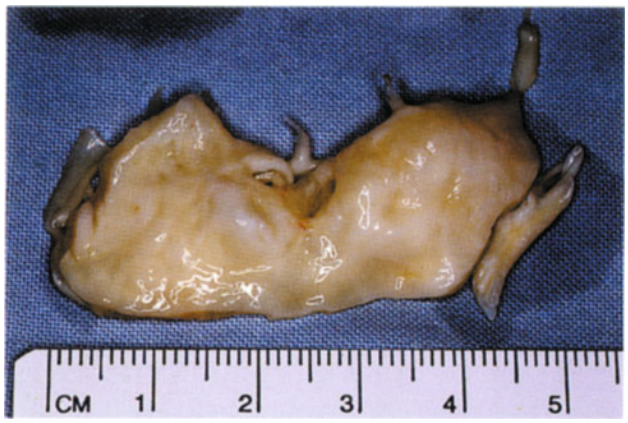


FIG. 9.5. A mitral valve repair always requires generous resection of the prolapsed leaflet to avoid systolic anterior motion (SAM) of the mitral valve

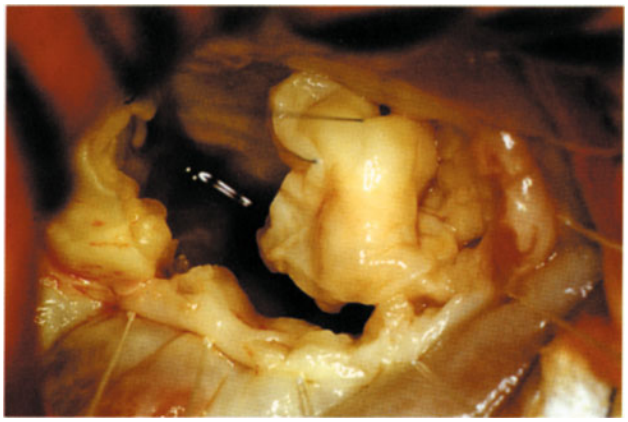


FIG. 9.6. Posterior leaflet reconstruction with leaflet-to-annulus approximation after leaflet sliding plasty

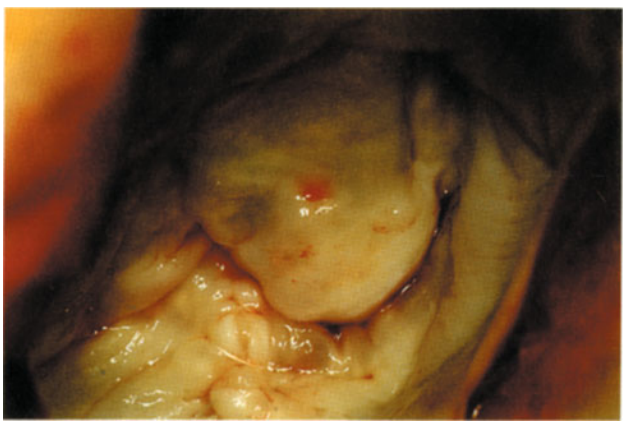


FIG. 9.7. The length ratio of the anterior leaflet to the posterior leaflet (3:1) needs to be considered in order to avoid development of SAM

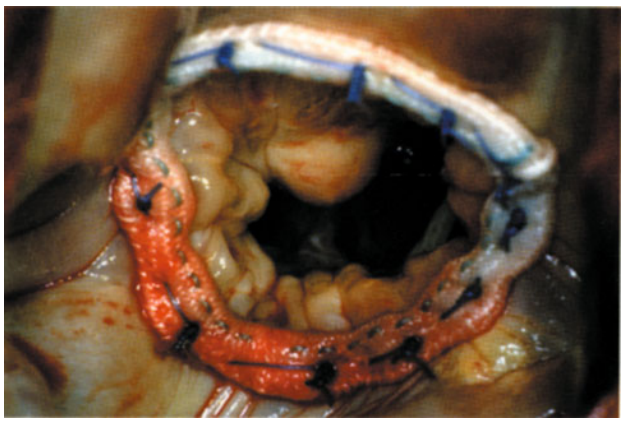


FIG. 9.8. After annulopasty ring implantation and proper estimation of (3:1) length ratio, SAM will not occur

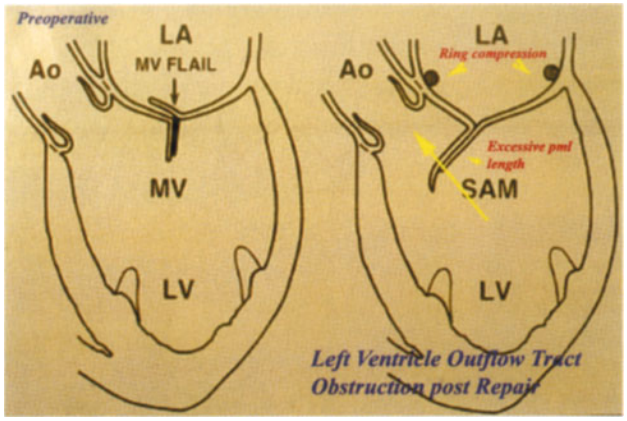


Fig. 10.1. SAM arises due to the excessive length of the anterior and/or posterior leaflet after valve reconstruction. The latter blocks the left ventricular outflow tract, especially after implantation of too small a composite annuloplasty ring

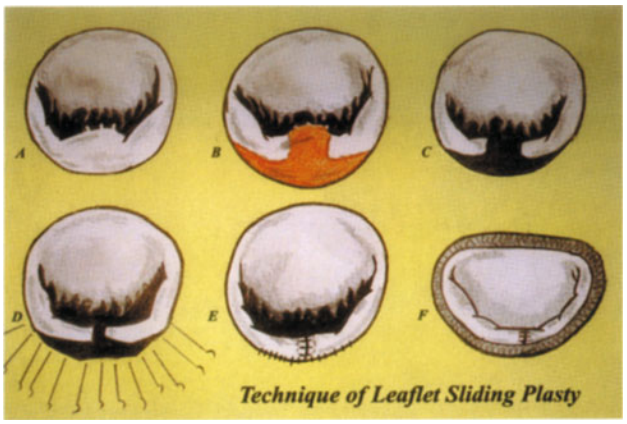
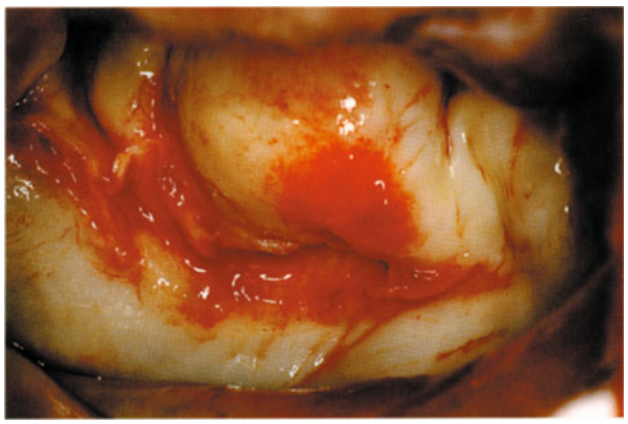


Fig. 10.2. This schematic demonstrates technique of leaflet sliding plasty



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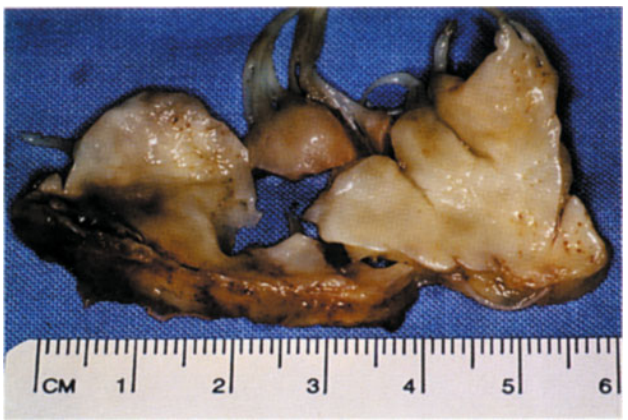
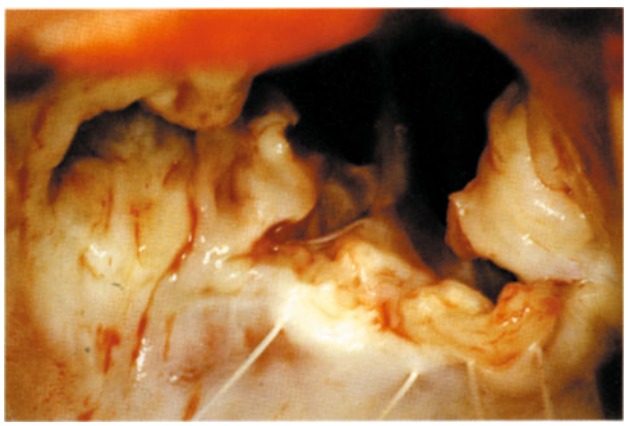


10.4

Figs. 10.3 and 10.4. After extensive removal of infectious tissue of the posterior leaflet and annulus



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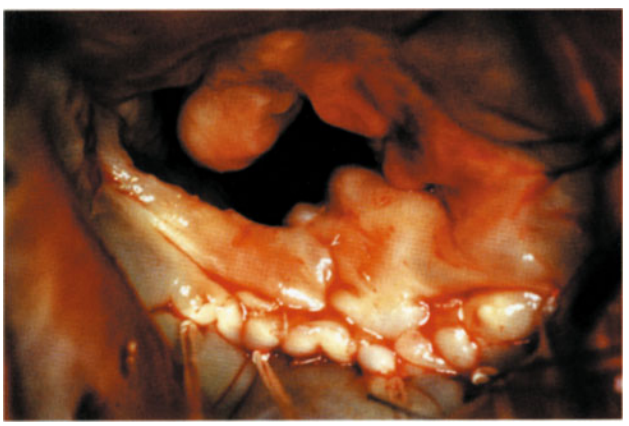


10.6

Figs 10.5 and 10.6. Show the surgical method of leaflet-annulus re-coaptation

is maintained and given a length ratio of 3:1 between the anterior and the posterior leaflet, there is no increased risk of SAM after implantation of an annulopasty ring (Fig. 9.8). *The technique of leaflet sliding plasty has been illustrated in detail in* (Fig. 10.2).

Using the *leaflet sliding plasty* technique and knowledge of the pathophysiology of SAM after repair, this hemodynamically significant complication can be avoided intraoperatively (Fig. 10.1–2). In addition, a surgeon skilled in this repair technique will be able to preserve the mitral valve after extensive resection of infectious tissue or after resection of calcification of the posterior annulus and the posterior leaflet (Fig. 10.3–6).

Leaflet sliding plasty has two special advantages: 1. – By utilization of this technique it is possible to preserve the valve after extensive resection of infectious tissue or after resection of calcification of the posterior annulus and the posterior leaflet. 2. – Additionally, this technique has the exceptional advantage of avoiding systolic anterior motion of the mitral valve after valvuloplasty (especially in Barlow's disease).

Leaflet sliding plasty

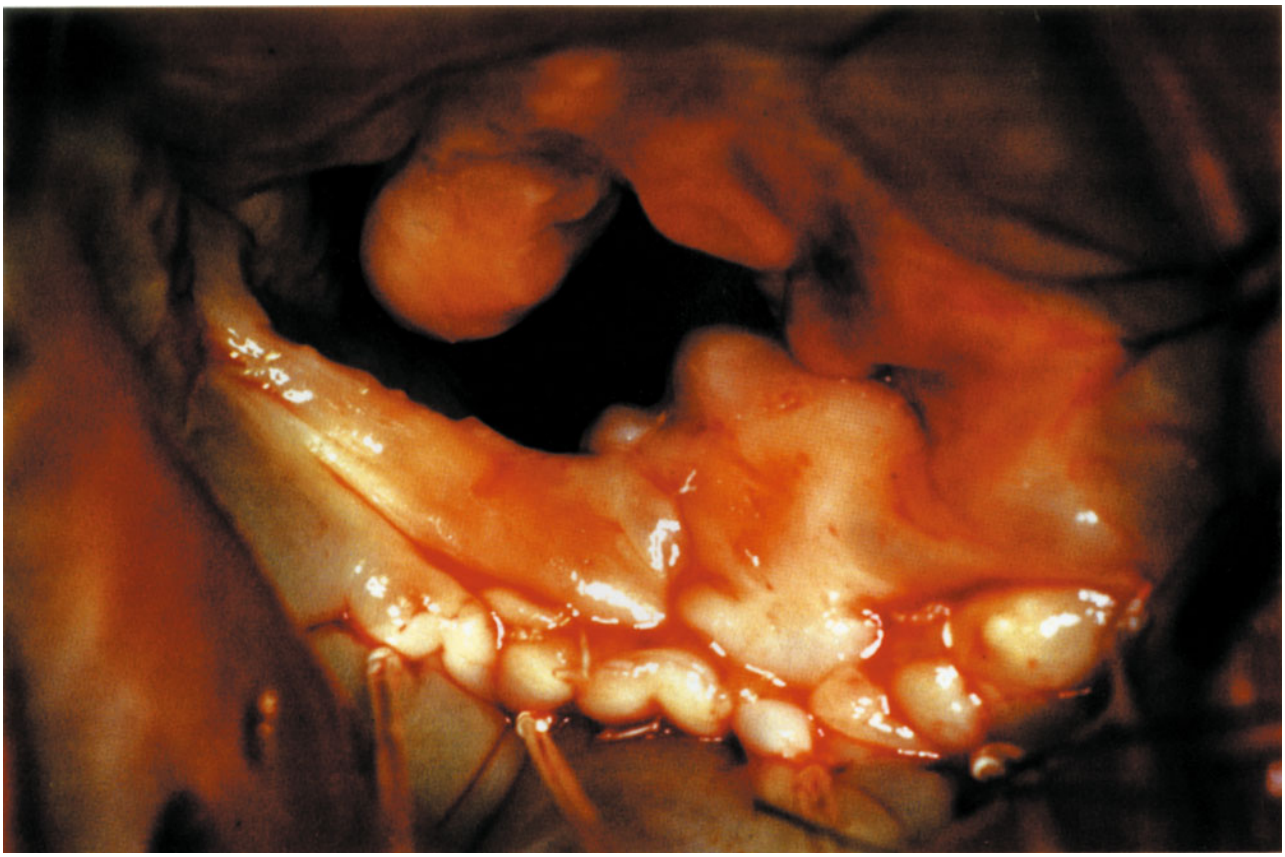


FIG. 10.6 (enlargement). After successful performance of sliding plasty

6 MITRAL VALVE SURGERY FOR COMPLICATIONS OF ENDOCARDITIS (Figs. 11–21)

Endocarditis is *defined* as microbial infection of the endothelialized surface of the heart. Vegetation, ulceration, perforation, dissection, abscess or rupture may occur after inefficient treatment [26].

Acute endocartits is usually caused by highly invasive organisms such as Staphylococcus aureus, streptococci, Haemophilus influenzae, Pseudomonas aeruginosa, Neisseria meningiltdis, or N. gonnorrhoeae. These infections may occur on either normal (up to 30% of patients having no identifiable underlying valvular lesion) or diseased valves because of the virulence of these organisms and their ability to adhere to the surfaces of valves. Bacteria that produce dextran appear to have greater adhesive properties, as do those organisms that produce components of extra cellular matrix such as fibronectin.

The pathogenesis of endocariditis requires several steps: (1) endothelial damage, (2) development of nonbacterial thrombotic vegetation, (3) adherence of circulating organisms to the vegetation, and (4) establishment and proliferation of infection in the vegetation. The importance of hemodynamic factors on the development of endocarditis is reflected in the relatively consistent locations of vegetation in endocarditis. In aortic regurgitation, the ventricular surface of the aortic valve and the chordae tendineae of the mitral valve are most commonly affected. In most cases of acute endocarditis, preexisting valvular disease has led to endothelial damages of the valve.

As a rule, acute endocarditis results in regurgitant lesions with rapid destructions of the valve. Infective endocarditis may irrespective of the underlying cause of the regurgitation. Although rheumatic heart disease and primary mitral valve prolapse are the most common pathologies encountered, papillary muscle dysfunction, usually result of myocardial ischemia, may also predispose to infective endocarditis.

Patients with pure or dominant mitral or aortic stenosis may develop increased stenosis of their valves during the course of infective endocarditis as a result infected thrombus or vegetations – so called Oslerian endocarditis. Surgery is indicated once theses changes or deterioration of clinical symptoms have been detected because the lesions are invariably hemodynamically severe. Systemic embolic in patients with embolic strokes resulting from infective endocarditis have a higher mortality are known.

Infection may occur as a primary or secondary process. Secondary infection results from infectious aortic valve by direct extension or via a jet of regurgitant blood. The usual cause of insufficiency is destruction of the leaflet or rupture of chordae. Less commonly, regurgitation may result from immobilization and retraction of the posterior leaflet when vegetation between the posterior leaflet and the ventricle heal. Following establishment of the infected vegetation on a leaflet, local spread of infection may occur. This generally occurs from the low pressure side of the cusp (atrial side for the mitral valve, ventricular side for the aortic valve) to the opposite side, then to other cusps (through contact or "*kissing*" lesions), and finally may extend to involve the chordae tendineae of the atrioventricular valves. This local spread of infection leads to acute regurgitation either through direct destruction of valve tissue, rupture of chordae tendineae in the atrioventricular valves, or, in aortic valves, by eroding cusp support when infection is near its commissural attachment and leading to prolapse of the leaflet. As vegetations grow in size, they may directly cause regurgitation by preventing proper closure of the valve. Further extension of the infection may occur into ventricular septum, resulting in septic myocarditis, abscess formation, and heart block if the conduction system is involved. The mycotic aneurysms formed by myocardial extension may rupture from the mitral valve ring into

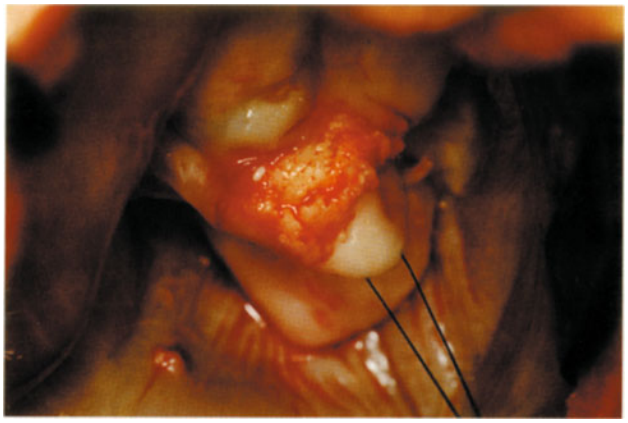


FIG. 11.1. Ulceration after endocarditis of the anterior leaflet

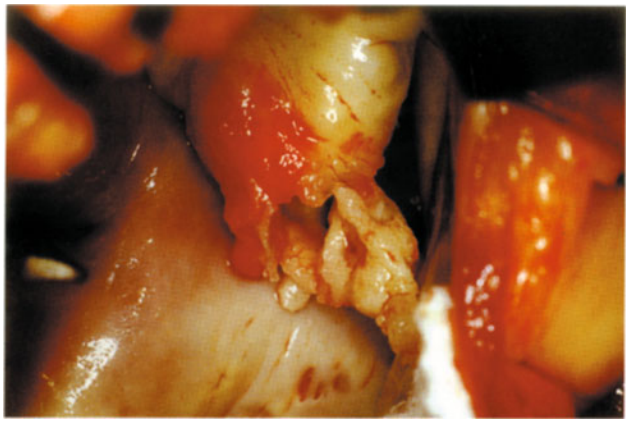


FIG. 11.2. Perforation and rupture as a complication after endocarditis

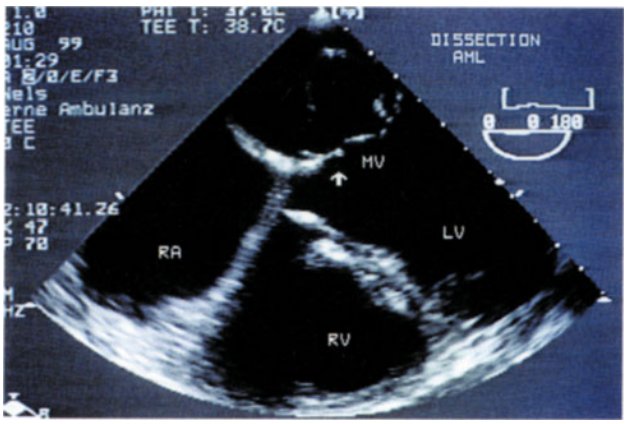


FIG. 11.3. The arrow on the 4-chamber TEE view indicates the dissection of the anterior leaflet

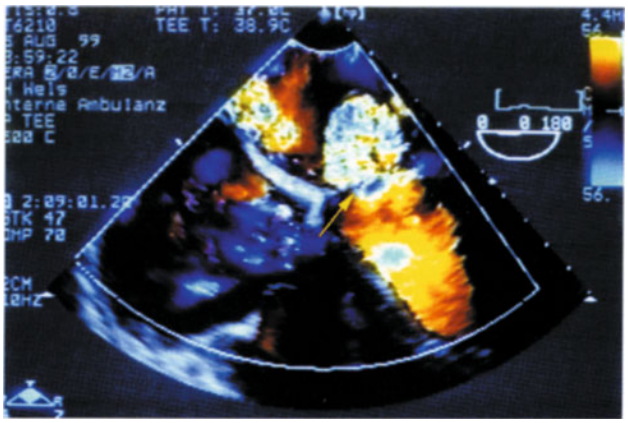
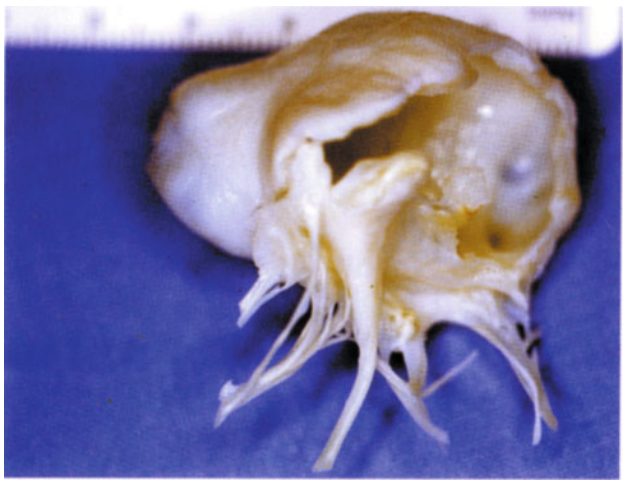


FIG. 11.4. Color Doppler reveals severe mitral regurgitation



11.5

11.6

FIGS. 11.5 and 11.6. Photographs demonstrates dissection at the coapting edge of the anterior leaflet and multiple perforations

the pericardium, or from the aortic root and adjacent septum into the right ventricle, the pulmonary artery, or the right atrium.

These complications are well depicted by TEE imaging. TEE aids not only in preoperative planning but also guiding surgery intraoperatively (Figs. 11–21).

Experience has shown that a *dissection* of the anterior leaflet can easily be detected on the TEE views: morpholic abnormalities in the valve and associated regurgitant jets are seen pointing out the pathology (Fig. 11.3–4).

Leaflet *perforations* can only be localized after careful echo analysis. Two-dimensional images show interruptions in leaflet anatomy; Color Doppler displays the site and mechanism of regurgitant jets. Single or multiple jets, often eccentric, are seen. Proximal flow convergence zone often point to the precise site of perforations (Fig. 12). It has been well-documented that TEE is highly sensitive in the detection of *vegetations*, their site and size (Figs. 15–16).

Figure 12.1, 2 is an example of post-endocarditic perforations in the anterior and posterior leaflet. Preoperatively, the perforations were confirmed on the color Doppler TTE apical longitudinal section as two narrow excentric jets. This was useful for informing the patient preoperatively and in surgical planning.

Figure 12.3–6 shows a post-endocarditic *ulceration* in the anterior leaflet on the TEE modified long-axis view with two different regurgitation jets on color Doppler. The *main stream* was due to the coaptation gap, while the narrow jet was presumed to be the result of a severe ulceration defect in the anterior leaflet. The complex valve pathology seen on the TEE was confirmed intraoperatively.

It is important not only to detect a wide regurgitation flow or main backward flow on color Doppler but also to perform an exact analysis of unusual jet lesions or *partial flows, which may be a conclusive indication of complex pathologies.* Precise analysis of the relations of different echo signals has implications in terms of preparation for surgery and for selecting a suitable surgical method [12]–[16].

Mitral and aortic regurgitation are the most common lesions to be exacerbated by infective endocardits and to subsequently result in *heart failure*. Heart failure is the most important cause of death in with infective endocarditis. Many investigators have demonstrated that early surgical intervention in native valve endocarditis improves survival if moderate or severe heart failure is present regardless of the activity of the infection. Potentially, infective endocarditis is correctable by surgery; this should be performed

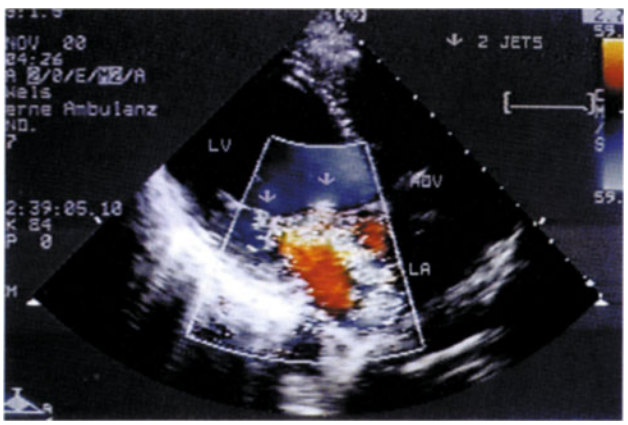


FIG. 12.1. Preoperative color Doppler TEE apical longitudinal image, demonstrates two small excentric jets (arrows) indicating perforations localized at the anterior and posterior leaflet

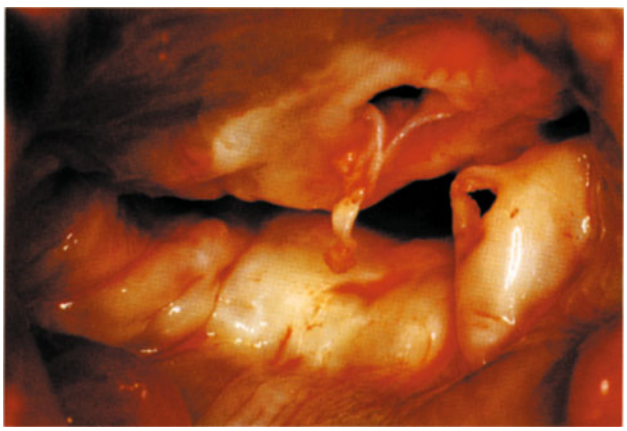


FIG. 12.2. Intraoperative view, perforations in the anterior and posterior leaflet with vegetations are visualized

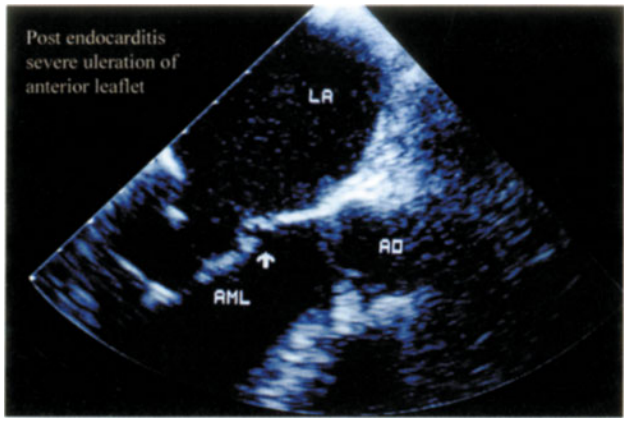


FIG. 12.3. The arrow shows post-endocarditic ulceration in the anterior leaflet on the TEE (modified long-axis)

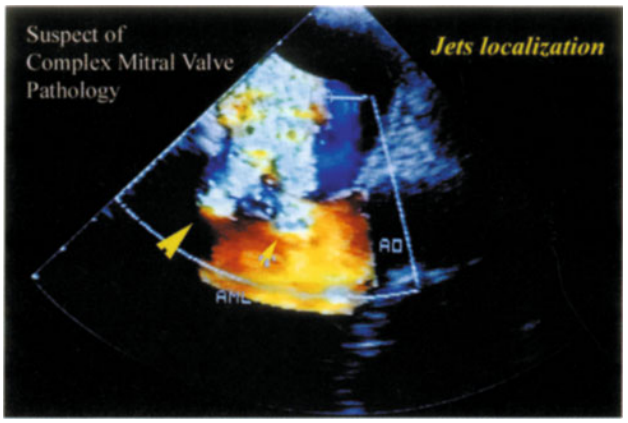


FIG. 12.4. Suspicious complex valve pathology with two different jet directions on color Doppler. The main stream is caused by the coaptation cap and the narrow jet, by a perforation in the anterior leaflet

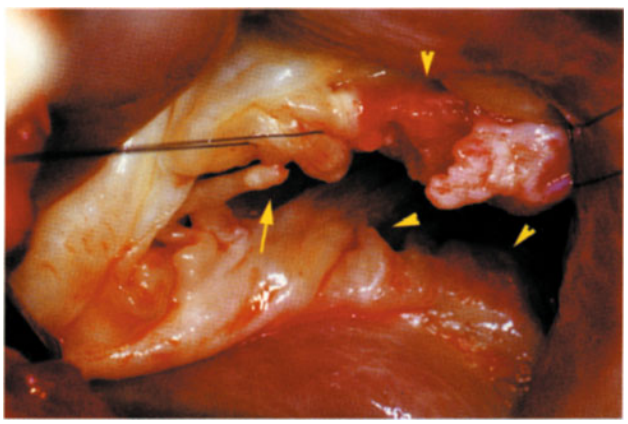


FIG. 12.5. The pathology of severe ulceration of the anterior leaflet with concomitant chordal rupture and elongation of P1 segment are seen. An ulceration of P II segment was also identified intraoperatively

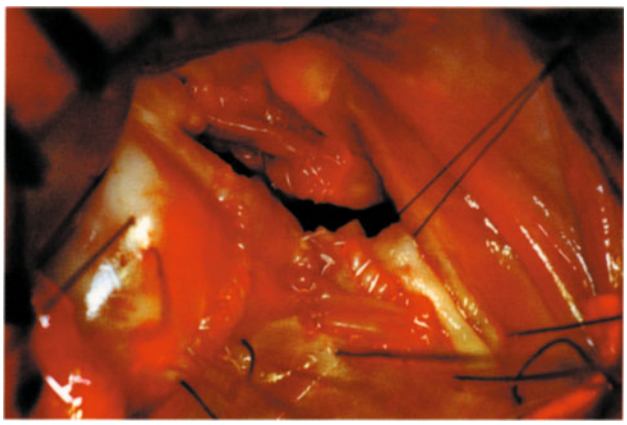


FIG. 12.6. Valvuloplasty, anterior and posterior leaflet replacement with treated autologous pericardium and P I approximation after extensive excision of infected tissues

too early rather too late on the whole. If a patient dies of heart failure before surgery, then the judgement of the cardiologist was invariably at fault. Another important and generally accepted indication for surgery is uncontrolled infection, defined as bacteremia or fungemia (or signs of sepsis) after a variable number of days of appropriable antimicrobial therapy or infection with a microorganism for which no effective therapy is available. A clear-cut example is fungal endocarditis, in which antifungal chemotherapy can relatively ineffective and valve sterilization with medical treatment alone is almost impossible. The presence of certain etiologic microorganisms, such as Staphylococcus aureus, or relapse after apparent medical cure suggest relative ineffectiveness of antimicrobial therapy, has been considered by most authorities to be an absolute indication for early valve surgery. The presence of vegetations on echocardiography has also

been correlated with an increased risk of subsequent congestive heart failure and therefore with the need for surgery.

Fairly mild edema resulting from acute-onset that is acutely produced, such as from rupture of a chorda tendinea, may result in pulmonary edema, which is not necessarily a reflection of true left ventricular failure and should respond well to medical therapy. Severity of the mitral regurgitation is easier to assess after the pulmonary edema has subsided, and assessment can usually be accomplished by clinical examination alone. Surgery often may not be required immediately or can be postponed for months.

Once aggressive approach to surgical management is indicated, there should be no conflicting concerns in conservative operations for mitral valve endocardits. Preservation of the subvalvular apparatus is necessary to maintain left ventricular function. Mitral valve repair is both feasible and desirable in these patients. Repaired valves are less thrombogenic than mechanical valve replacements. Unlike bioprostheses, they have excellent long-term durability. However, accidental retention of infected valve tissue could lead to postoperative endocarditis, particularly if a prosthetic annuloplasty ring is applied.

6.1 The principle of surgery in active infective endocarditis

The principle of surgery in active infective endocarditis is removal of all infective endocarditic tissue and all vegetations on valvular structures and repair of damaged structures (Figs. 13–16).

In some cases, the required extent of resection is so extensive that repair becomes practically impossible (Fig. 14.1–3). For repair, the ideal material is local *autologous pericardial* tissue, which should be used along with the tissue remnants to model a functional valve (Fig. 13.2, Fig. 14.4, Fig. 15.6). Patients with endocarditis who have developed an abscess in the mitral annulus or in the myocardium close to the valve require innovative reconstruction techniques. The resection and treatment of such abscess cavities is only successful when extensive debridement and reconstruction of the wall is undertaken, and by suturing an autologous patch of pericardium that is fixed in a sufficiently stable manner to the ventricular musculature as well as beyond the annulus in the atrial wall (Figs. 15–16) [20], [23], [27], [28].

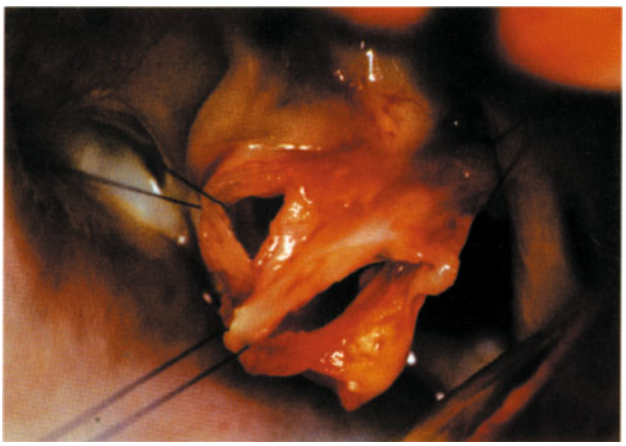


FIG. 13.1. Visualization of perforation of the anterior leaflet after acute bacterial endocarditis that had caused severe mitral regurgitation

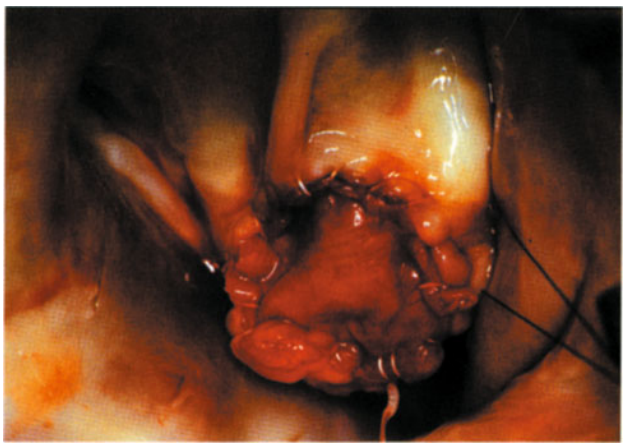


FIG. 13.2. Valve reconstruction with autologous pericardium as leaflet substitute

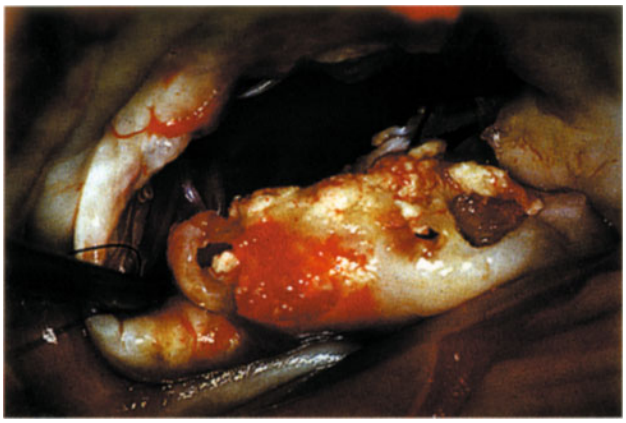


FIG. 14.1. Severe bacterial endocarditis of the posterior leaflet

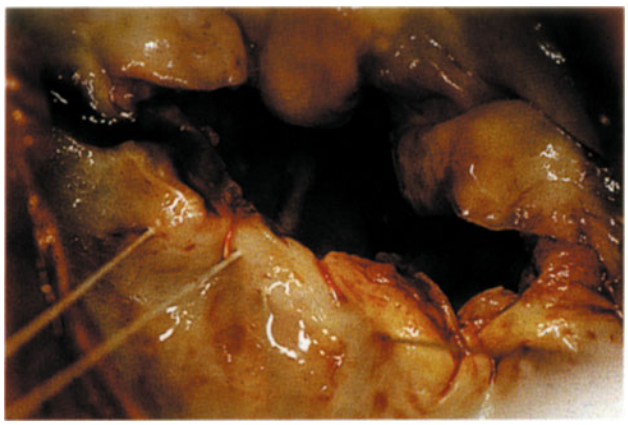


FIG. 14.2. Extensive resection of the infectious tissue

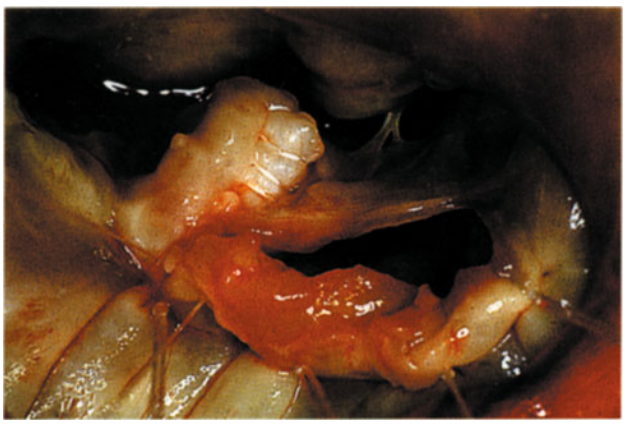


FIG. 14.3. Large gap between leaflet surface remnants and the annulus, which renders primary reconstruction endeavors impossible

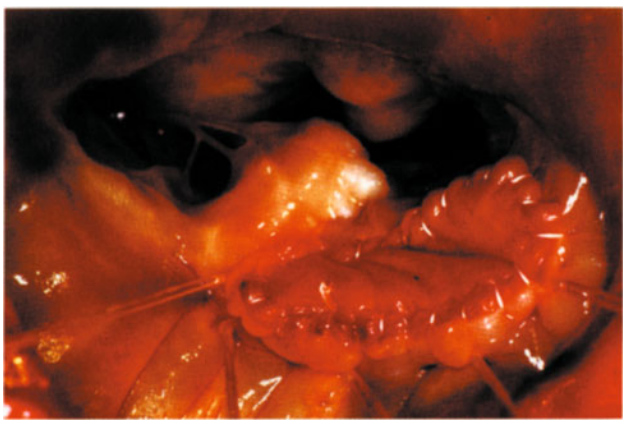


FIG. 14.4. Valvuloplasty with a pre-treated autologous pericardial patch as substitute for the posterior leaflet

6.2 Timing of the operation, extracardial complications

The question of *timing the operation* in active infective endocarditis is also an important issue. In this difficult patient population, the right time point of surgery is an essential element of the modern surgical strategy. It is important to avoid unnecessary delay in order to prevent serious complications as well as to minimize surgical mortality and postoperative morbidity. Longer the delay, greater is the tissue damage and more difficult is the operation [30], [31], [32].

Urgent valve surgery *is indicated in endocarditis patients with severe regurgitation or obstruction causing hemodynamic instability.* Other indications include fungal endocarditis, persistent bacteremia, presence of abscesses or fistulae, resistant infection exhaustion of all antibiotic measures or recurrent endocarditis after primarily successful antibiotic treatment. Early surgery should be performed in all cases of intracardial spread of infection and rupture of the chordae tendinae or papillary muscles, rupture of the sinus valsalvae or the ventricular septum and in patients with heart blocks caused by involvement of the conduction system in

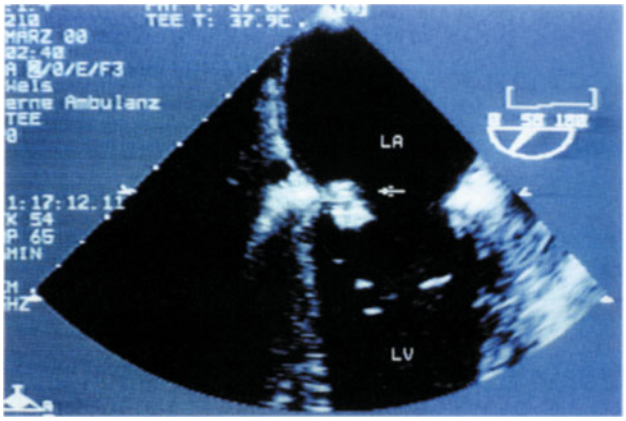


FIG. 15.1. The arrow on the TEE 4-chamber view indicates a large vegetation (larger than 10 mm)

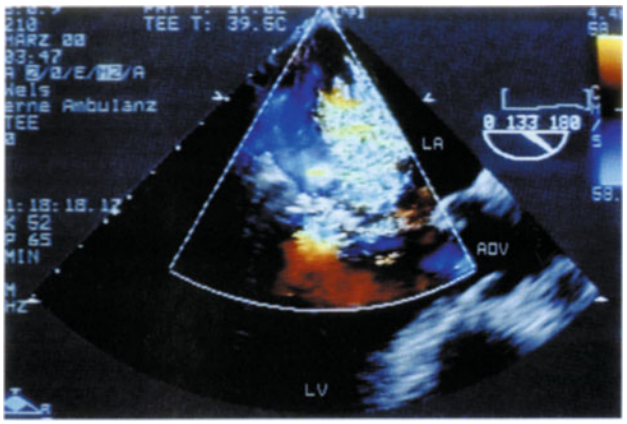


FIG. 15.2. Severe mitral regurgitation (stage IV) on long-axis TEE view is demonstrated

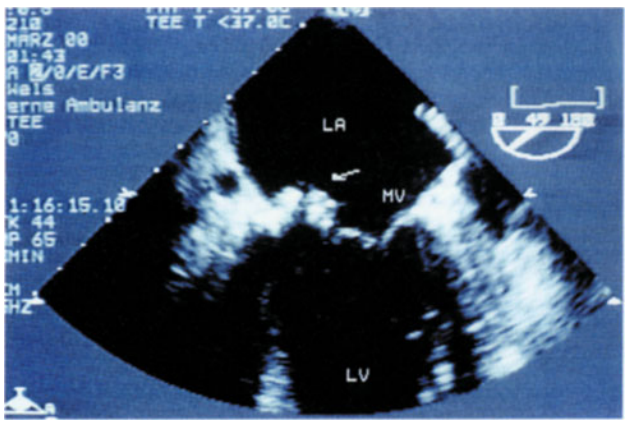


FIG. 15.3. The vegetation on TEE is described to be extremely mobile and fragile. The abscess has spread to the atrium, has destroyed the posterior leaflet (P3 segment), is extending to the annulus and further into the adjacent myocardium

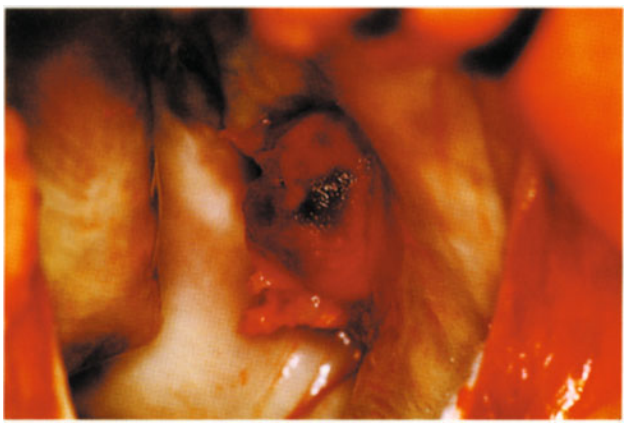


FIG. 15.4. The vegetation – as seen in the preoperative echo – has destroyed the posterior annulus and the adjacent posteromedial commissure as well as parts of the atrial tissue

infection. All these complications are well identified by echocardiography [10], [13], [14], [30].

Patients who have had an embolism at least once previously also need urgent surgical intervention, especially in cases of proven vegetations on echocardiography or *when vegetations are found to be persistently growing* [13]. Surgery is needed in cases of large infective spread of vegetation (more than 10 mm) on echocardiography. In the patient shown in Figs. 15–16, the vegetation was extremely mobile and fragile on TEE. The abscess had spread up to the atrium, destroyed the posterior leaflet (the P3 segment) and projected beyond the annulus into the adjacent myocardium (Fig. 15.1–4).

An established severe **extracardial complication** like pulmonary embolism is *an immediate indication for surgery* in the presence of active infective endocarditis, as the prognosis is unfavorable in the absence of surgical intervention [28], [30]–[32].

Active endocarditis with rapid growth of vegetation in the posterior annulus and the subvalvular apparatus, up to a size of 20 mm, and

intracardiac spread of the same due to infection in the septal trabeculae, was found in a 37-year-old female patient (Fig. 16.1–2). The patient had previously had a *peripheral embolization* with *septic purulence* of the skin, septic spondylitis, subdural hematoma and bleeding in the basal ganglia. Although all drug therapies had been exhausted, the patient had persistent fever. The ECG showed an AV block. Immediately before surgery the patient develpoed sudden dyspnea, tachycardia, and hypoxic. Massive pulmonary embolism was suspected. It was confirmed on CT

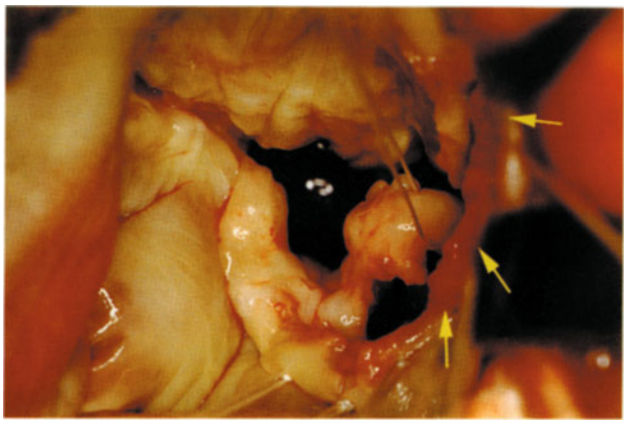


FIG. 15.5. After complete debridement of the infectious tissue, one half of the posterior annulus,the surrounding atrium and the corresponding chordae tendinae could be preserved for reconstruction

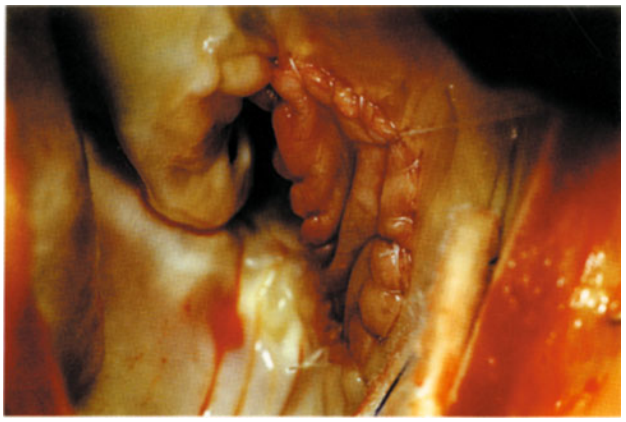


FIG. 15.6. Valve reconstruction with autologous pericardium as substitute tissue from the commissure to the middle leaflet scallop, to cover the annulus and the adjacent neighborhood myocardium, followed with reimplantation of separated chords

Mitral valve repair in a patient with extensive complications of endocarditis

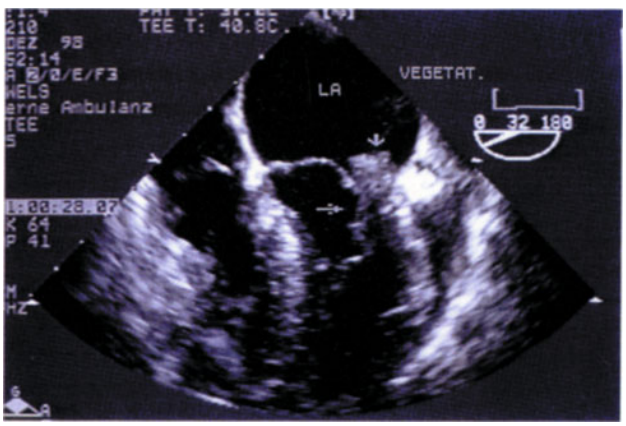


FIG. 16.1. Active endocarditis with fast growth of vegetation in the posterior annulus and subvalvular apparatus (size of more than 20 mm on the 4-chamber view TEE (arrows))

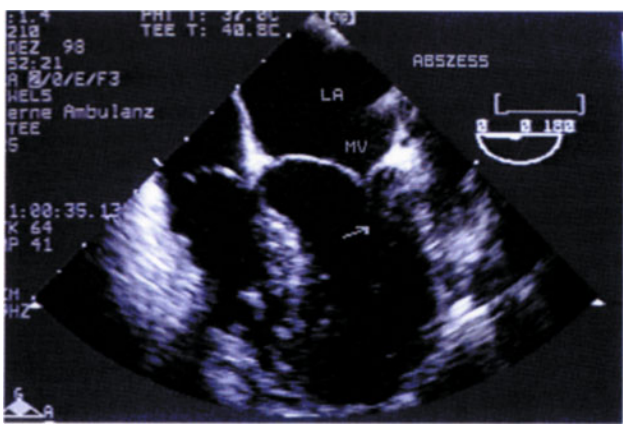


FIG. 16.2. Evidence of intracardiac spread of infection to the myocardial trabeculae (arrow). The (female) patient had previously suffered peripheral embolization with septic pustules into the skin,septic spondylitis, subdural hematoma and bleeding into her basal ganglia. Although drug therapy was extensive, the patient remained febrile. The ECG showed an AV block. Immediately before surgery, the patient had sudden dyspnea, and severe hypoxia refractory to medical therapy

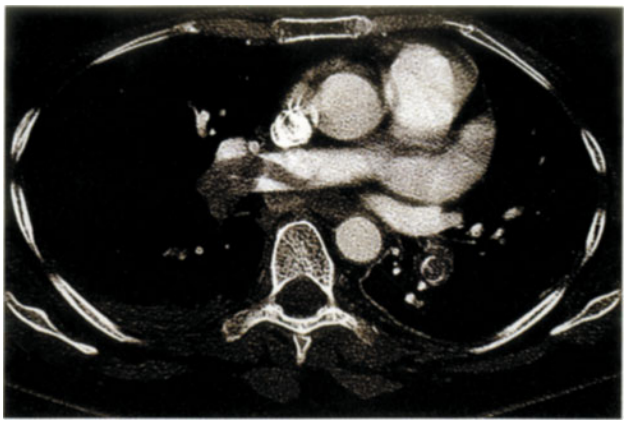


FIG. 16.3. Massive pulmonary embolism from the pulmonary trunk to the periphery was confirmed on a computed tomography scan CT

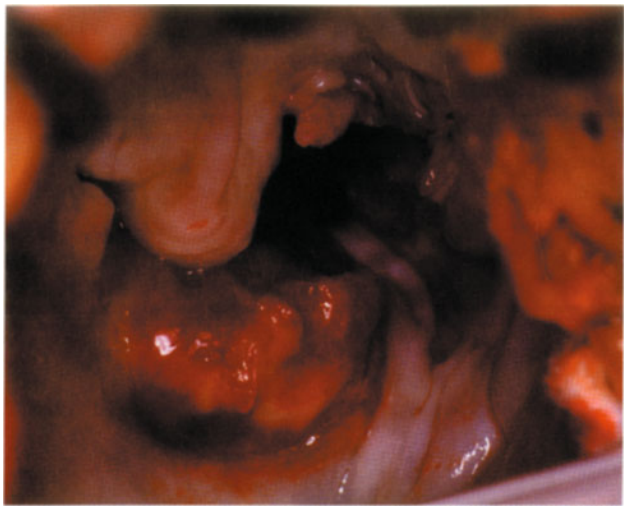


FIG. 16.4. Vegetations, damaged leaflets and annulus and extention of infection into the adjacent myocardium are seen

FIG. 16.5. Excised thrombi after pulmonary thrombembolectomy before mitral valve reconstruction

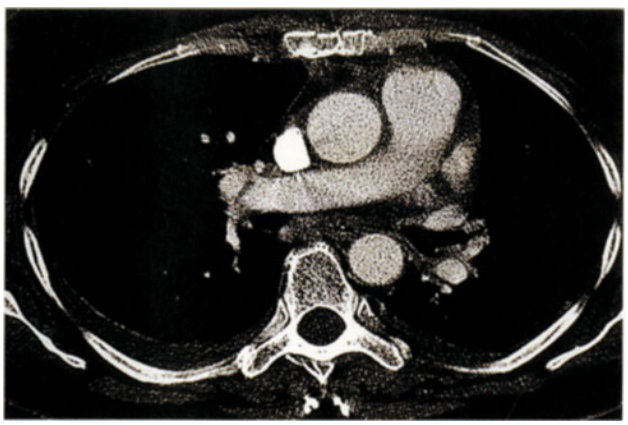


FIG. 16.6. The postoperative control CT before discharge demonstrates the success of the operation with complete recanalization of the pulmonary artery

in Fig. 16.3. *An immediate surgical intervention was the life-saving measure for this patient* (Fig. 16.6).

6.3 Destruction of commissures

In cases of infective endocarditis, **destruction of commissures**, regardless of the side that is affected, is always followed by severe mitral regurgitation. The dysfunction affects both leaflets simultaneously and is frequently accompanied by rupture of the paramedian chordae and the struct chordae (Figs. 17–20). Even the slightest strain is liable to cause decompensation in these patients within an unexpectedly short period of time. Therefore, the decision to perform surgery must be made at short notice in this setting [26]–[28], [33]. The *TEE* examination usually shows a flail leaflet or a prolapse of both leaflets. Color Doppler often reveals *an oblique, eccentric regurgitation jet* between the two leaflets (Fig. 17.1–2).

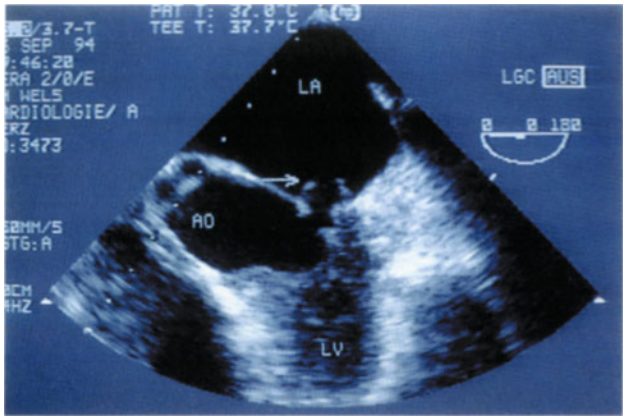


FIG. 17.1. The arrow on the TEE transverse section shows a flail leaflets preoperatively

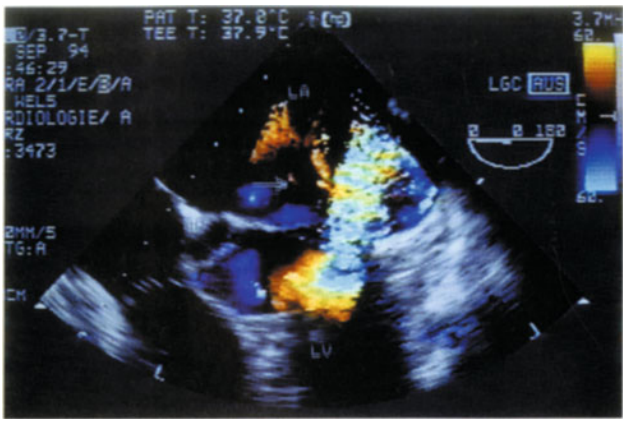


FIG. 17.2. An oblique regurgitation jet between the two leaflets, pronounced sideways, is a reliable sign of commissural pathology on color Doppler

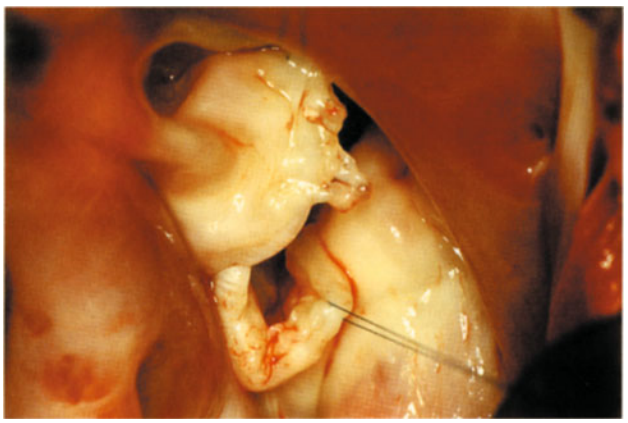


FIG. 17.3. Operative view demonstrates postendocarditic destruction of the anterolateral commissure with rupture of the paramedian chordae and the struct chordae

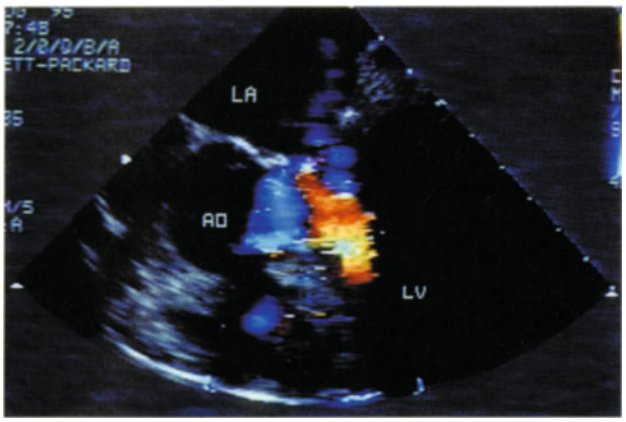


FIG. 17.4. After repair with rotation paracommissural sliding plasty, the TEE transverse section shows no regurgitation

The previously mentioned *rotation paracommissural sliding plasty* is suitable for such a complex commissural pathology, as it provides good long-term success [24] (Fig. 8.5–6, Fig. 17.4, Fig. 19.3–4). The diagnostic procedure should be complemented by a *TTE or TEE short-axis view of the mitral valve* in order to confirm that the selected surgical method is the appropriate one. The commissural gap between the anterior and posterior leaflet can be very precisely localized on short-axis views (Fig. 18.3, 4; Fig. 20.3, 4). In patients with an unclear commissural rupture, this diagnostic option should be used in every case. The short-axis view in the cross-sectional TTE is a simple and efficient method to evaluate commissural pathology [13].

Once the diagnosis has been established, a suitable method of reconstructing the commissure should be selected. Complete repair can even be performed in cases of extensive posterior leaflet perforation with involvement of the paracommissural segment of the anterior leaflet and the commissural annulus with multiple vegetations (Fig. 19.1, 2). After extensive segmental resection of both leaflets, the two leaflets can be re-coapted by employing the previously

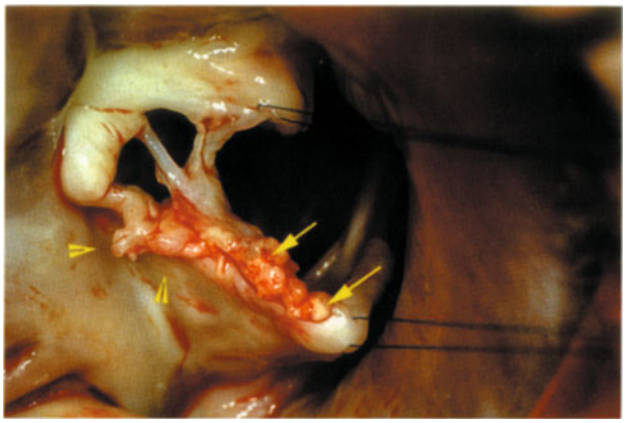


FIG. 18.1. Acute bacterial endocarditis with rupture of the entire anterolateral paracommissural chordae

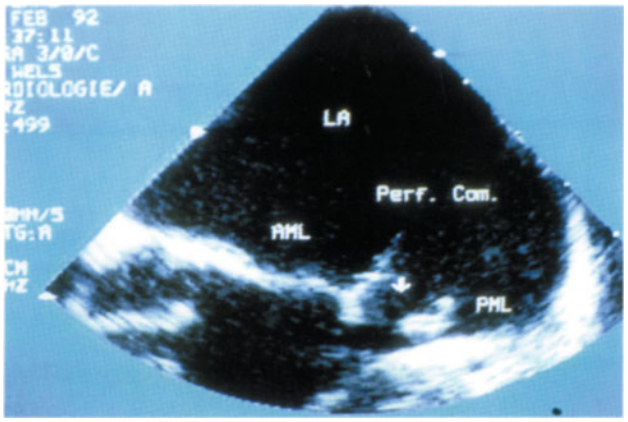


FIG. 18.2. The pathological problem, as indicated by the arrow on the preoperative TEE, raises suspicion of a commissural perforation with detachment of both leaflets

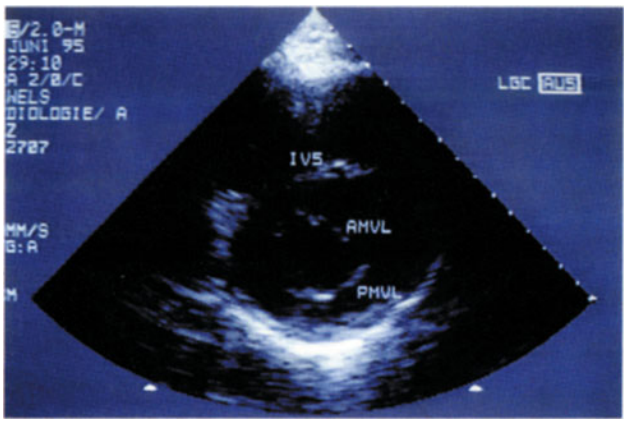


FIG. 18.3. For precise echo diagnosis and analysis of commissural pathology, TTE short-axis view examination is helpful to recognize the commissural gap between the anterior and posterior leaflet

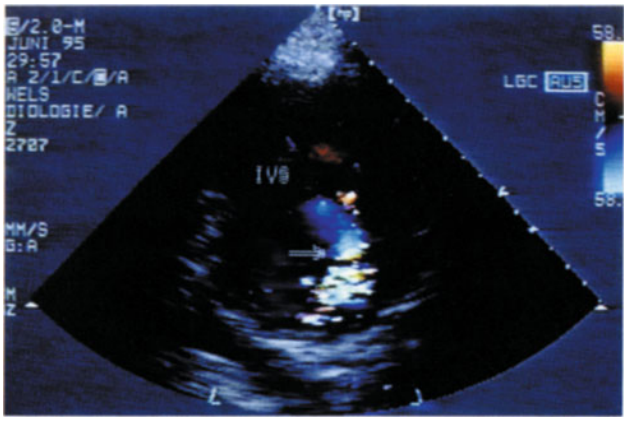
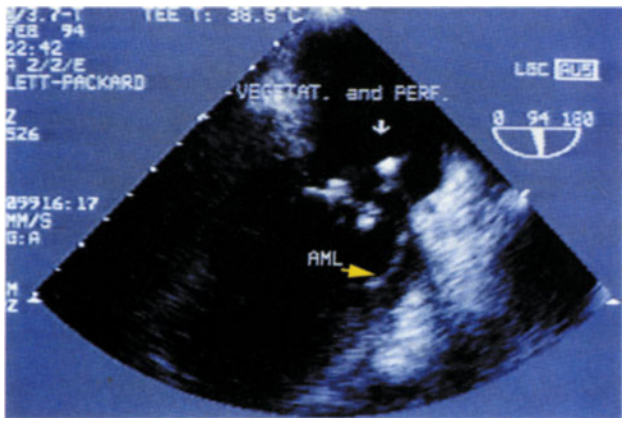
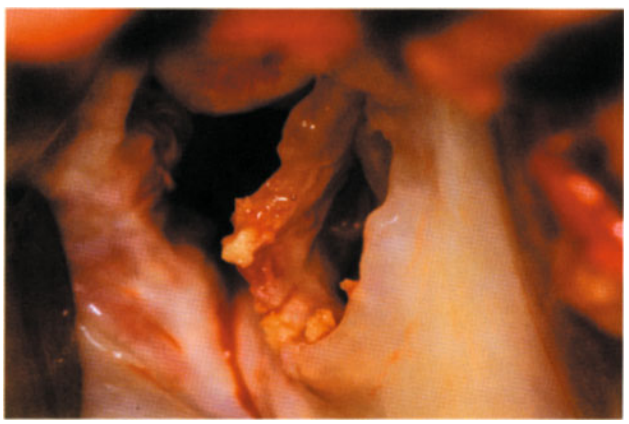


FIG. 18.4. Color Doppler displays mitral regurgitation at the site of the commissural gap



19.1



19.2

FIGS. 19.1 and 19.2. TEE (longitudinal view) and intraoperative photography show a large area of endocarditis with posterior leaflet perforation. Involvement of the paracommissural segment of the anterior leaflet and the surrounding annulus with multiple vegetations is also demonstrated

described method of rotation paracommissural sliding plasty. Patients with complex commissural pathologies should always be treated with a *composite annuloplasty ring*. A composite ring (Fig. 19.5) stabilizes the entire circumference of the annulus and should therefore be used especially after manipulation at the commissures or in their vicinity [16], [34].

For optimal treatment of various complex pathologies after endocarditis of the mitral valve, the *use of a patch* in valvular reconstruction often is the only solution, failing which the valve would have to be replaced [16], [19], [20], [22], [23]. Frequently-used patch operation techniques are the following: patch as substitute for the posterior leaflet, paracommissural extension patch plasty, posterior leaflet extension patch plasty, patch as substitute for the anterior leaflet, or as chordal replacement. In our patients, only autologous pericardial patches fixed in 0.62% glutaraldehyde-buffered solution have been used. The technique of fixation has been described on page 11 (4.2).

6.4 Patch as substitute for the anterior leaflet (Fig. 13)

In patients with perforation of the leaflet after active or healed bacterial endocarditis, partial leaflet replacement is performed. For prevention of renewed infection and correction of mitral insufficiency, a circular patch is employed to repair the defect after excision of the edge of the perforation and the infectious tissue. A continuous 7-0 PTFE suture is used for this purpose (Fig. 12.5, 6; Fig. 13.1, 2; Fig. 21.5, 6).

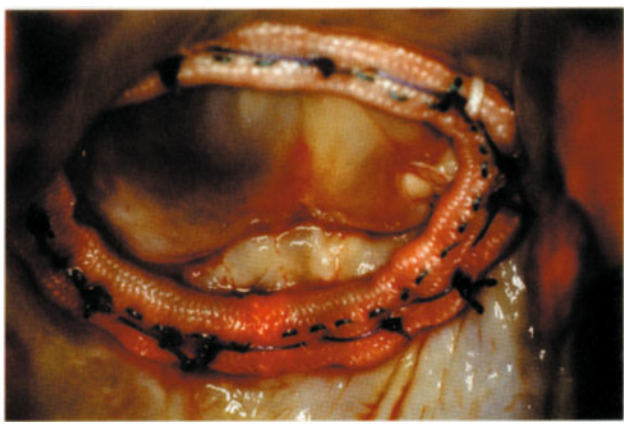
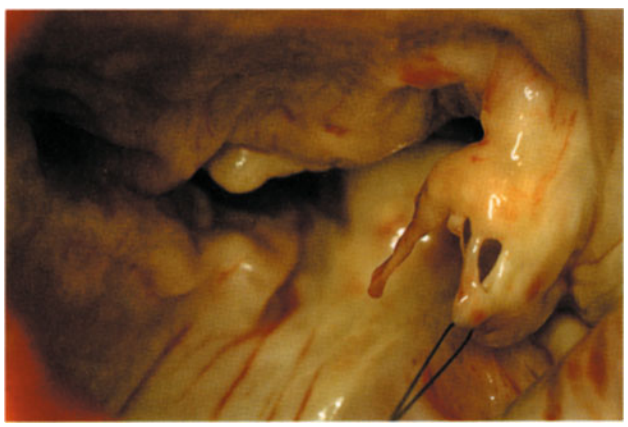
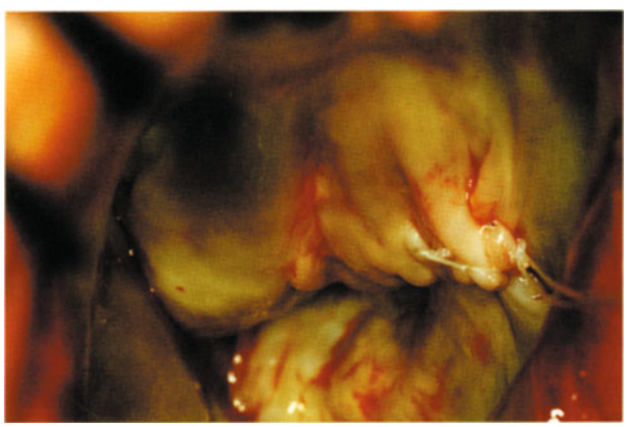


FIG. 19.5. In cases of commissural pathology, mitral valve repair is better performed with a composite annuloplasty ring. It should be noted that a ratio of 3/4 between the anteroposterior and transverse length should not be exceeded, in order to prevent the occurrence of SAM. Excellent coaptation of both leaflets with restored commissures and implantation of a composite ring are optimal prerequisites for the long-term success of valve reconstruction. After successful repair, the curved coapting closure line can be compared with the mouth of a "smiling valve"



19.3



19.4

FIGS. 19.3 and 19.4. After extensive segmental resection at the posteromedial commissure, good re-coaptation with neoformation of a posteromedial commissure is achieved with rotation paracommissural sliding plasty

6.5 Patch as substitute for the posterior leaflet (Figs. 14–15)

We have applied his technique in two groups: Group I were endocarditis patients with a destructive process in which extensive tissue resection was required; Group II were patients who had severe degenerative prolapse involving more than 2/3 of the posterior leaflet with mild to severe calcification that occasionally involved the posterior annulus. The patients in these two groups had pliable anterior leaflets. However, after removal of the posterior leaflet and part of the annulus, neither the standard technique with sliding annuloplasty nor the posterior leaflet folding plasty as described by Grossi [16], [35] was able to achieve reconstruction of a competent valve because of a lack of sufficient tissue. These valves were selected to perform repair with a pericardial patch. After complete excision of the infected area, removal of vegetations and abscesses or decalcification of the annulus, 5-0 U sutures of PTFE were made at the annulus. Tension at the U sutures permitted circular reduction of the size of the annulus. Tension at the leaflet remnants allowed for adjustment of the position of the leaflet towards the annulus. The small quantity of remaining leaflet tissue could not be connected to the annulus to form a single unit. The empty space between the leaflet and the annulus had to be bridged in order to achieve repair. The edges of the prepared strip of pericardial tissue were fixed to the annulus and to the leaflet tissue with a continuous 6-0 or 7-0 suture (Fig. 12.5, 6; Fig. 14.1–4; Fig. 15.5, 6).

6.6 Paracommissural extension patch plasty (Fig. 20)

In another patient the commissural area had to be filled with a crescent-shaped patch because

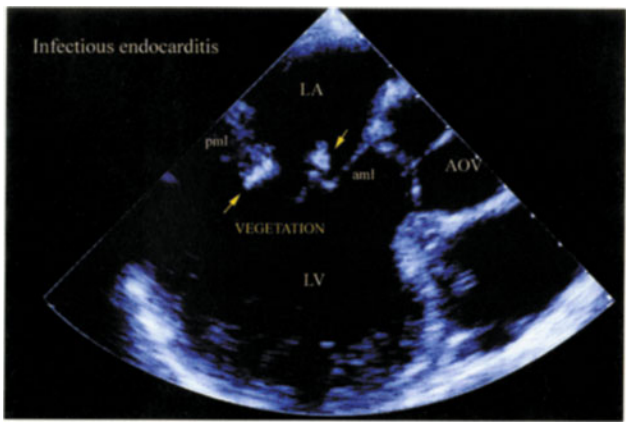
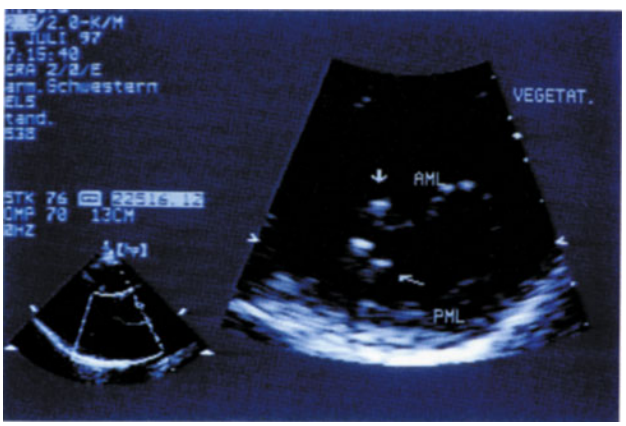
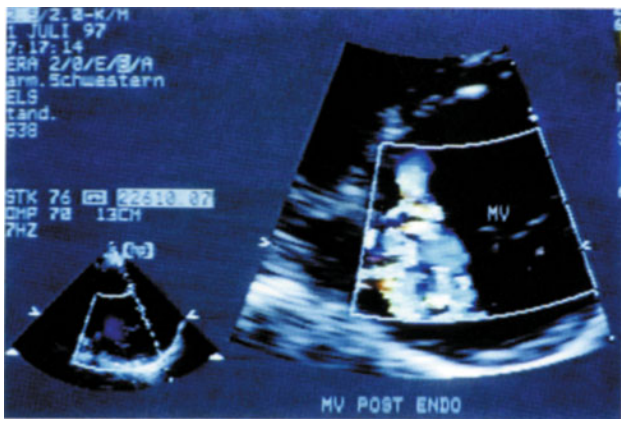


FIG. 20.1. Large vegetations in the anterior and posterior mitral leaflets in a patient with fungal endocarditis are seen on the preoperative TEE image



20.2



20.3

FIGS. 20.2 and 20.3. The short-axis view TTE is advantageous. Here it displays the severity of the main pathology at the posteromedial commissure, vegetation, tissue destruction and severe mitral regurgitation

of tissue shrinkage after rheumatic endocarditis, tissue destruction with perforation or colonization and vegetations as a result of bacterial processes, or even after anterior and posterior leaflet prolapse owing to ruptured commissural chordae. The pathological commissural tissue was first detached from the annulus. A patch was inserted by means of sliding annuloplasty in order to create an area of approximation at the two leaflet remnants. The surface of approximation functioned as a "neo-commissure" (Fig. 20.5, 6).

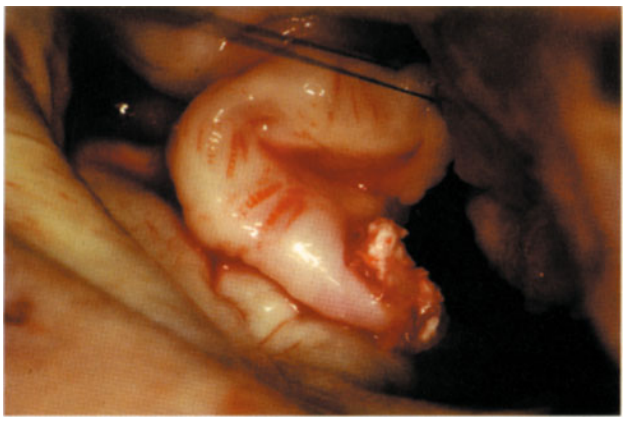
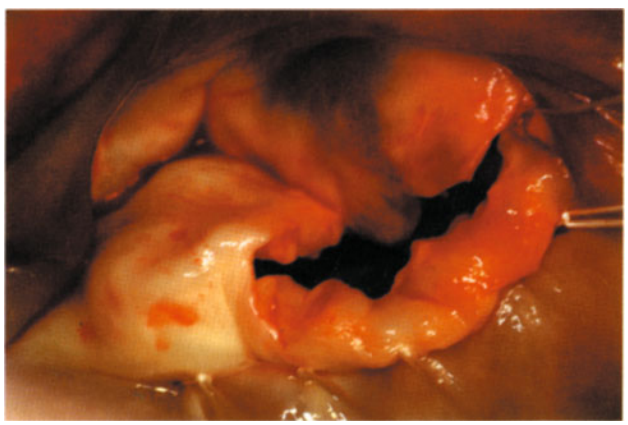


FIG. 20.4. Intraoperatively, cheese-like vegetations are noted in this patient with active fungal endocarditis

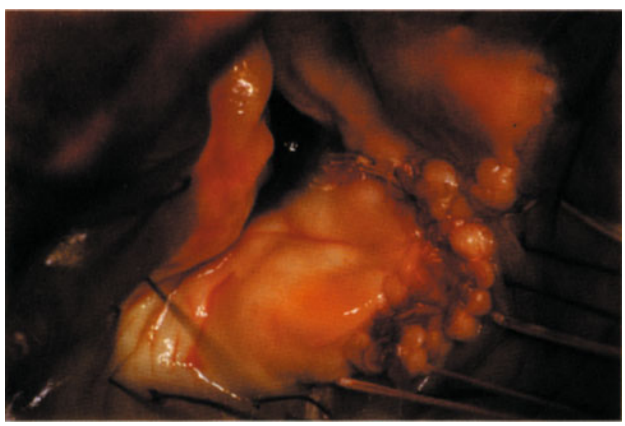
6.7 Mitral valve surgery in the presence of active, infective aortic valve endocarditis (Fig. 21)

In many cases, destruction of the mitral valve is caused by active infective aortic valve endocarditis (Fig. 21). The result is a so-called **jet lesion** secondary to the reflux of blood through the insufficient endocarditic aortic valve and its impact on the anterior mitral leaflet, which projects into the left ventricular outflow tract during diastole (Fig. 21.1, 2). Accordingly, such jet lesions are located in the middle of the anterior leaflet; they may range from trivial deposits of vegetation to an extensive perforation of the leaflet (Fig. 21.5) [27]–[29].

Using a *transaortic access* for reconstruction of the anterior mitral valve leaflet may even avoid additional tissue damage in the left atrium.



20.5



20.6

FIGS. 20.5 and 20.6. After extensive resection of posteromedial paracommissural parts of the anterior and posterior leaflet, a paracommissural extension patch plasty is performed with an autologous pericardial patch

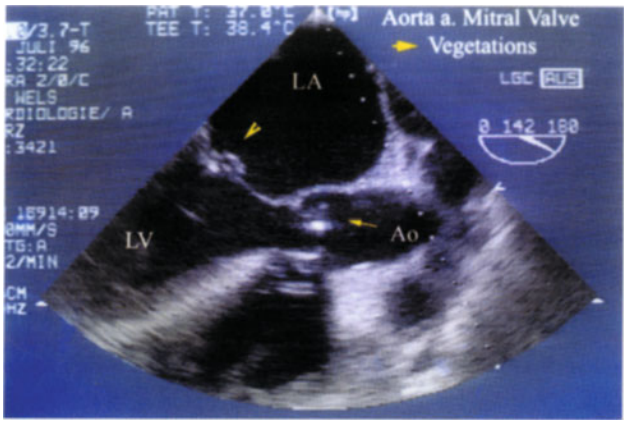


FIG. 21.1. Acute infectious endocarditis of the aortic and mitral valve; the arrows show deposits of vegetation on both valves in the TEE long-axis view

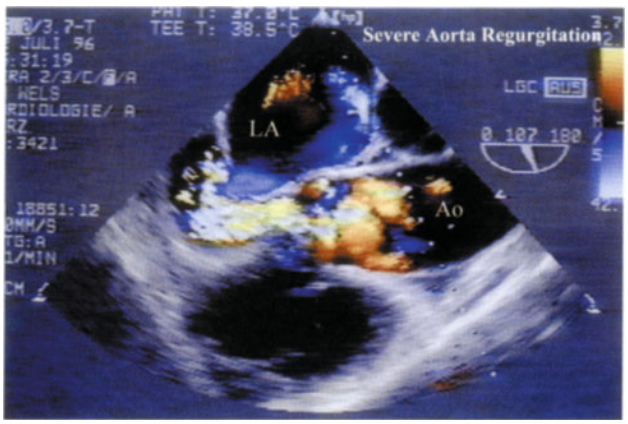


FIG. 21.2. The jet lesion arising from the reflux of blood through the insufficient aortic valve and its impact on the anterior mitral valve leaflet can be observed on color Doppler

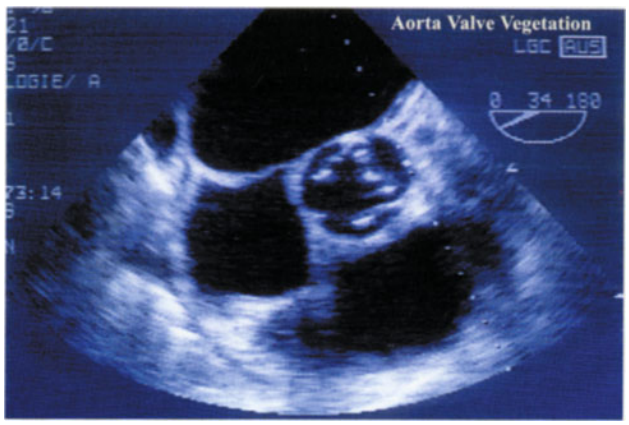


FIG. 21.3. TEE image of the aortic valve, the primary valves involved in endocarditis shows damages cusps

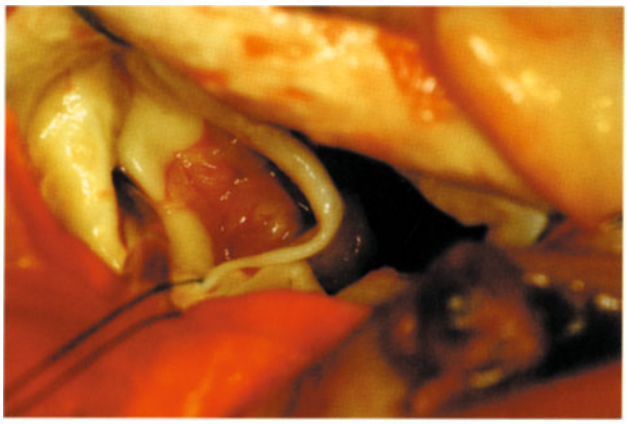


FIG. 21.4. Intraoperative visualization of vegetations in the left coronary sinus and below the aortic valve annulus

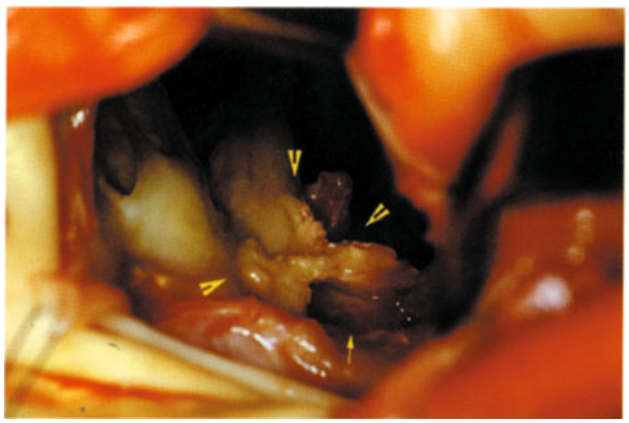


FIG. 21.5. Transaortic visualization of the jet lesion after removal of the aortic valve vegetations and the destroyed aortic valve leaflets. The thick arrows show vegetations and infection sites in the anterior mitral leaflet; the thin arrow points to the perforated anterior mitral valve leaflet

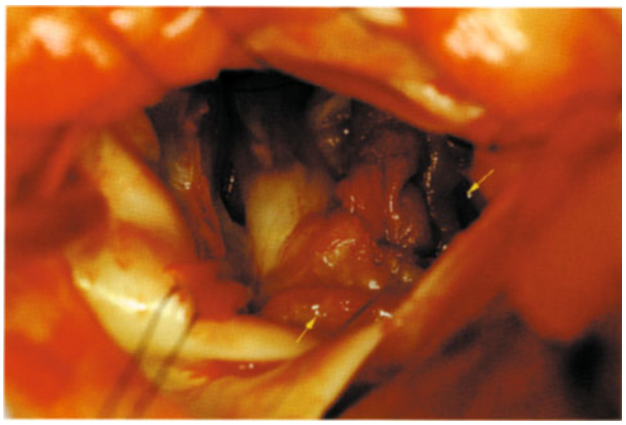


FIG. 21.6. Transaortic reconstruction of the anterior mitral valve leaflet with a pericardial patch as leaflet substitute

Mitral valve surgery for complications of endocarditis

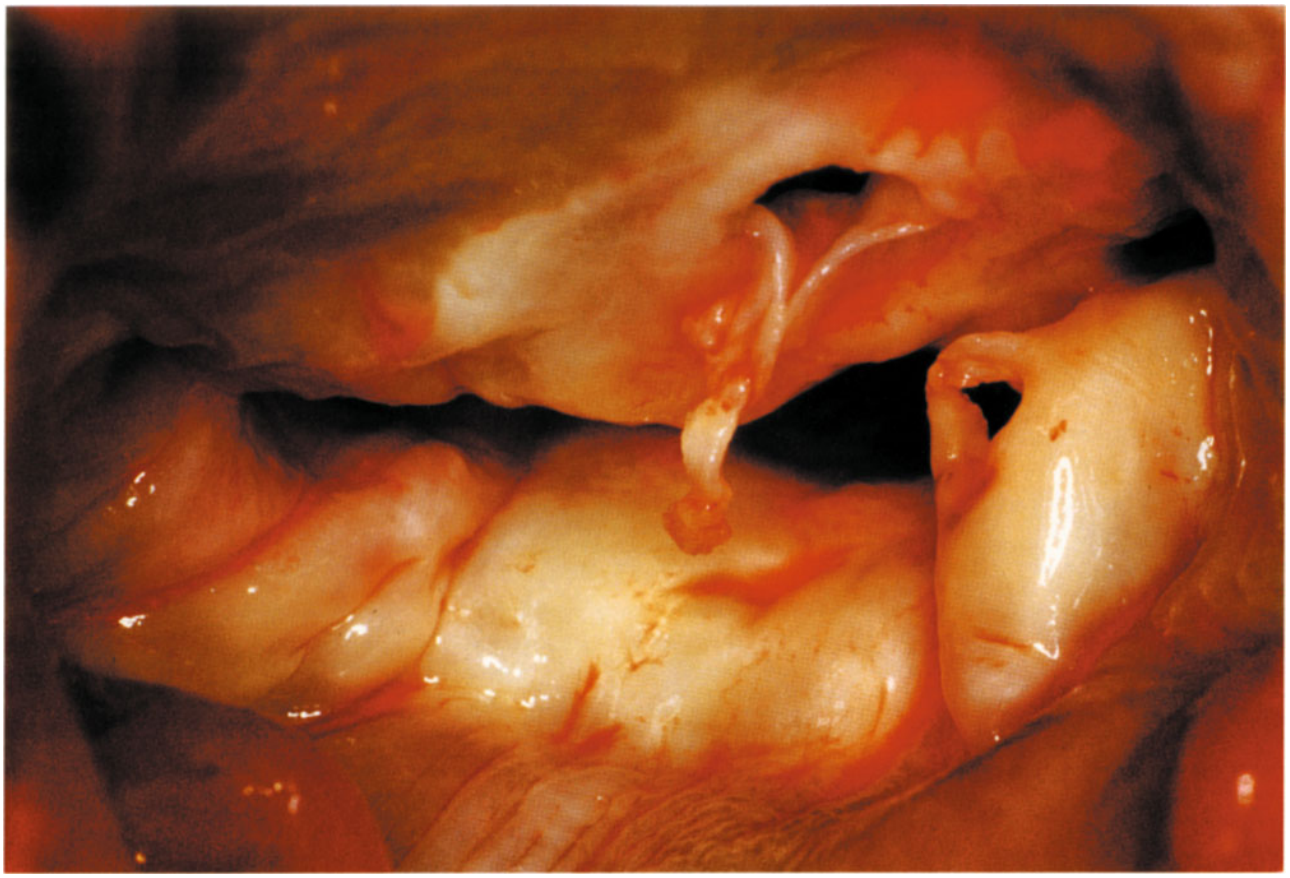


FIG. 12.2 (enlargement). Intraoperative view, perforations in the anterior and posterior leaflet with vegetations are visualized

Mitral valve surgery for complications of endocarditis

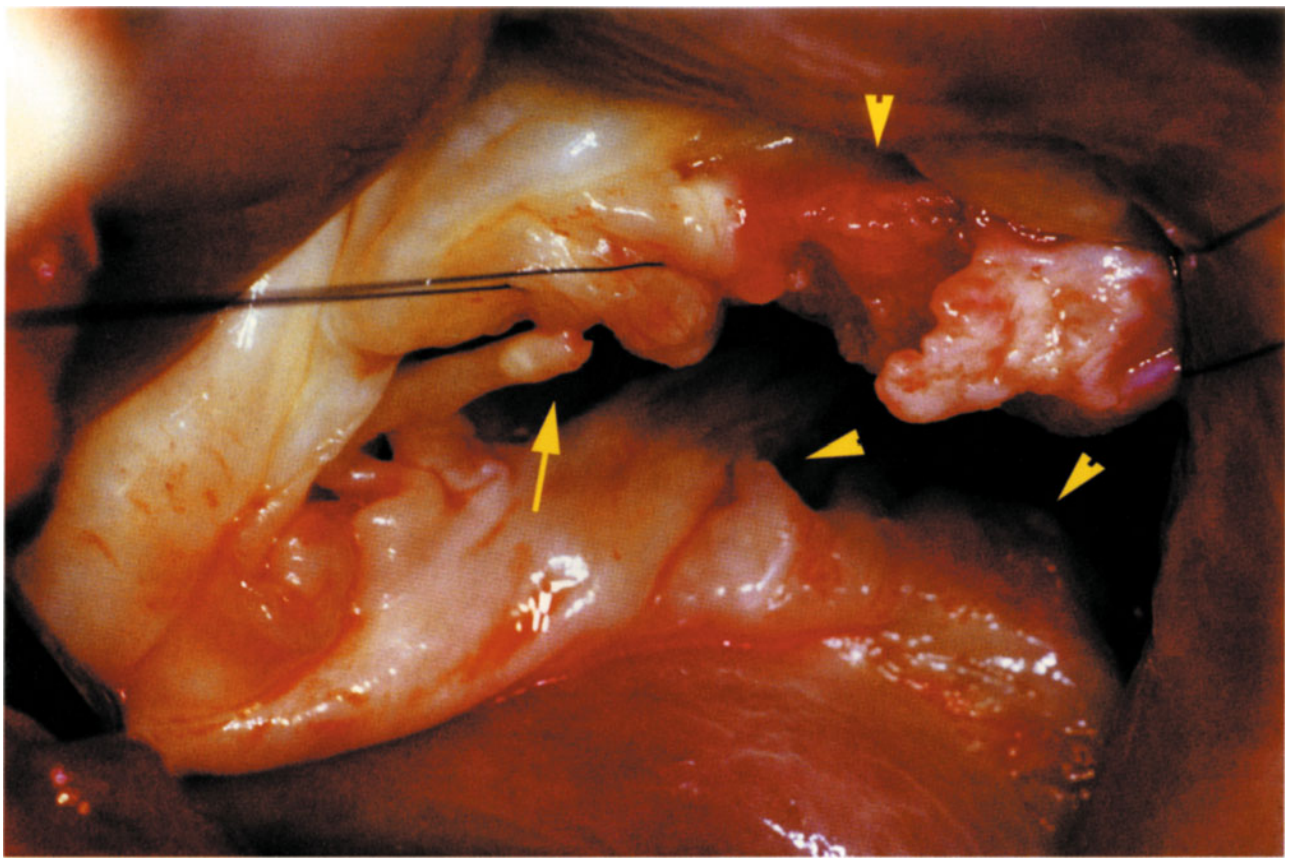


FIG. 12.5 (enlargement). The pathology of severe ulceration of the anterior leaflet with concomitant chordal rupture and elongation of P1 segment are seen. An ulceration of PII segment was also identified intraoperatively

Mitral valve surgery for complications of endocarditis

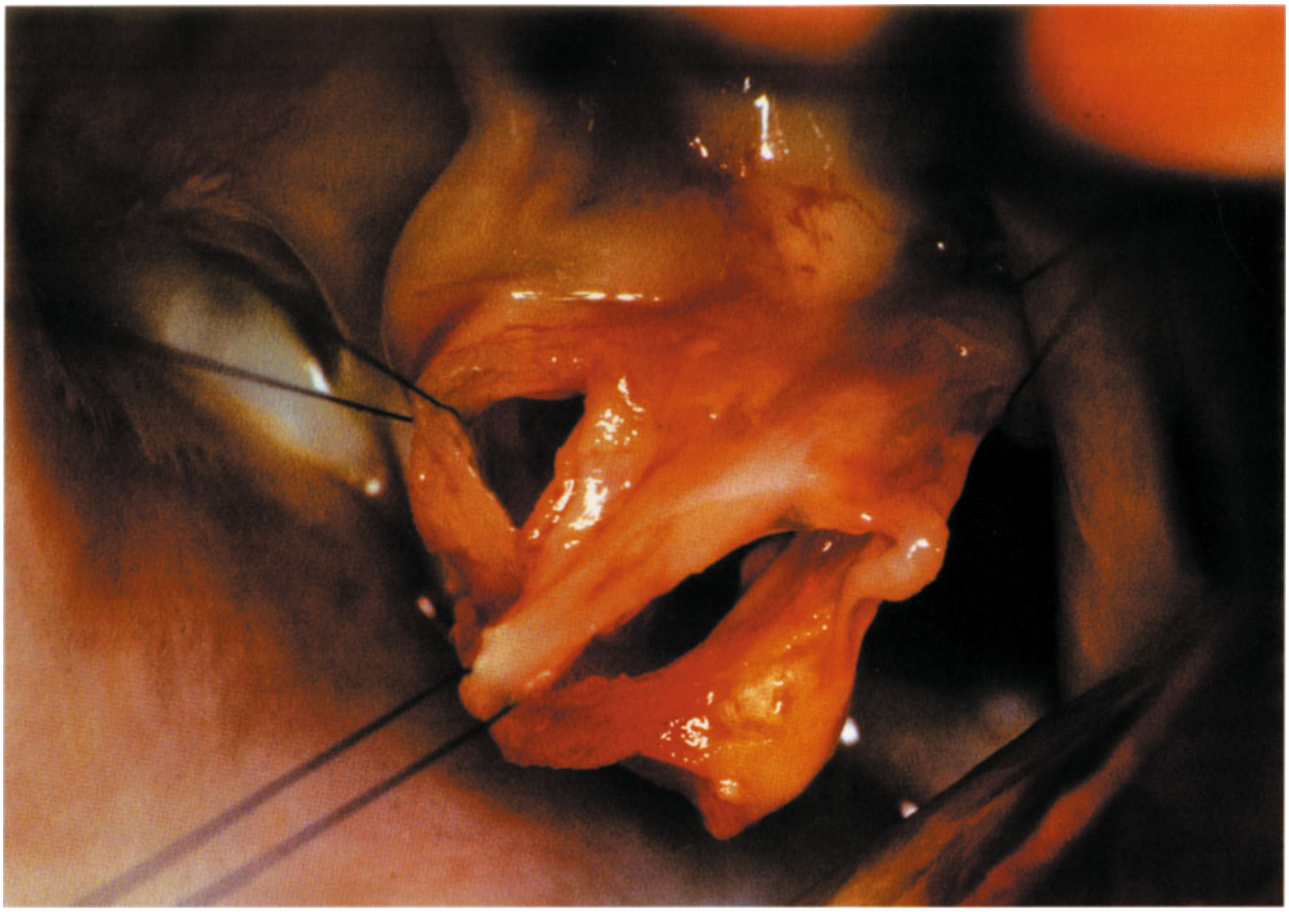


FIG. 13.1 (enlargement). Visualization of perforation of the anterior leaflet after acute bacterial endocarditis that had caused severe mitral regurgitation

The principle of surgery in active infective endocarditis

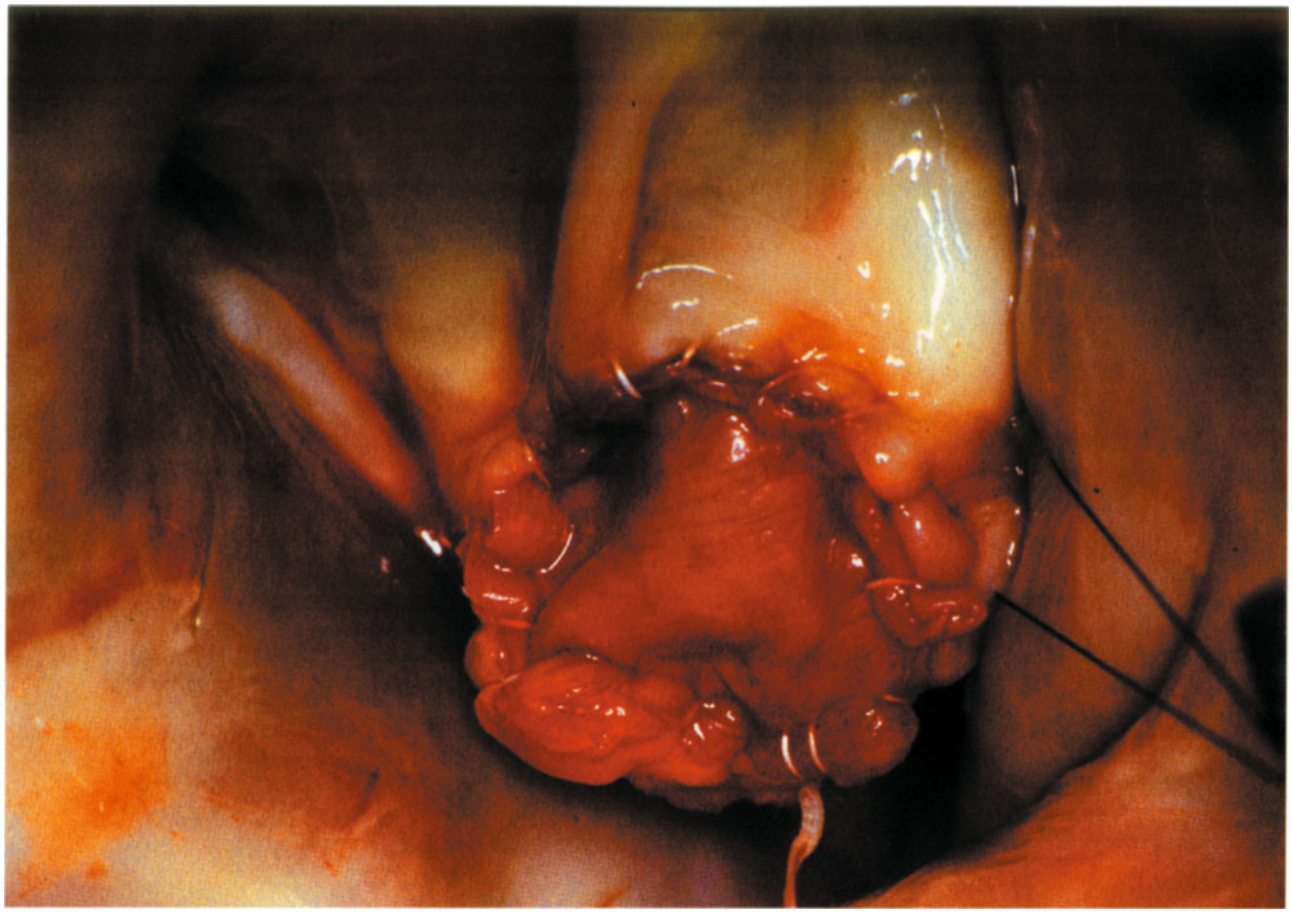


Fig. 13.2 (enlargement). Valve reconstruction with autologous pericardium as leaflet substitute

Mitral valve surgery for complications of endocarditis

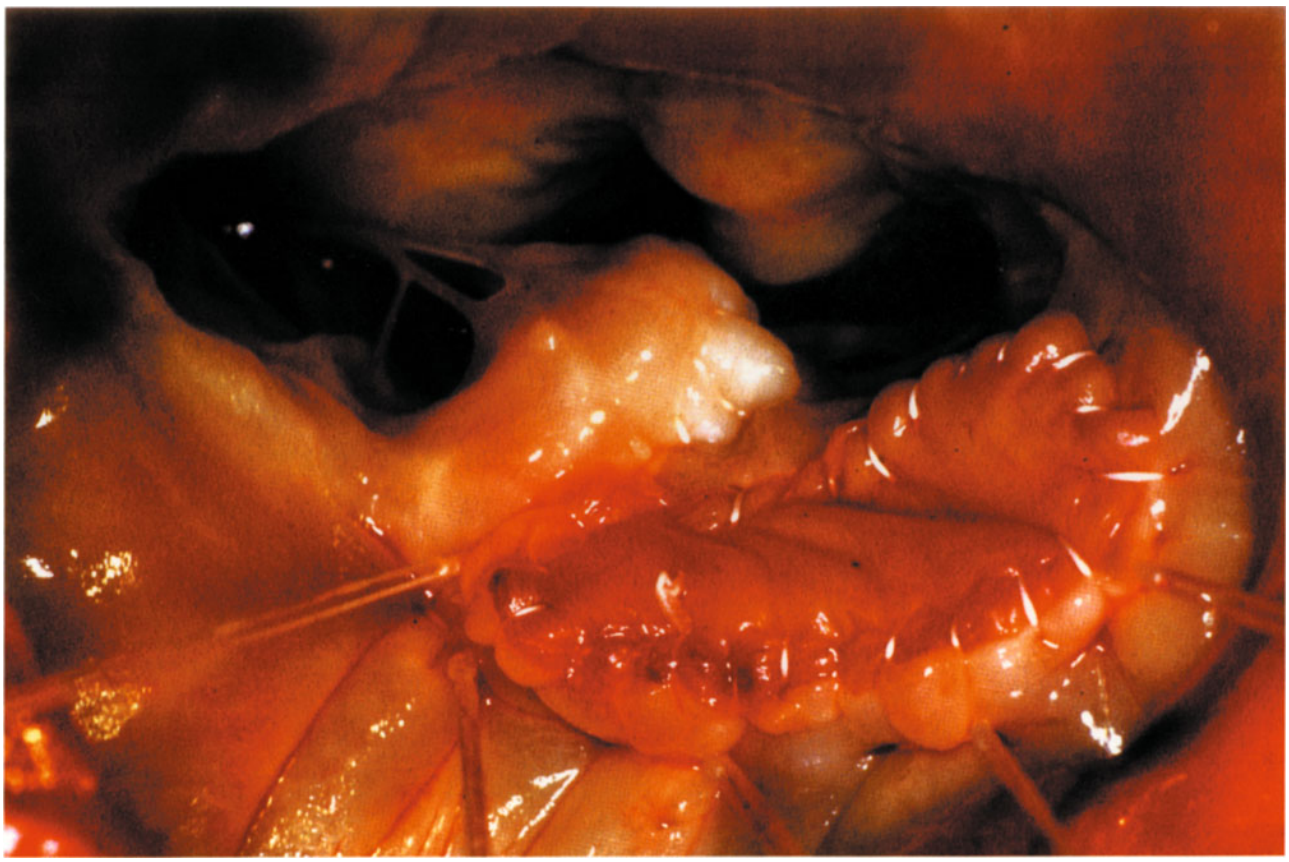


FIG. 14.4 (enlargement). Valvuloplasty with a pre-treated autologous pericardial patch as substitute for the posterior leaflet

Mitral valve surgery in the presence of active, infective aortic valve endocarditis

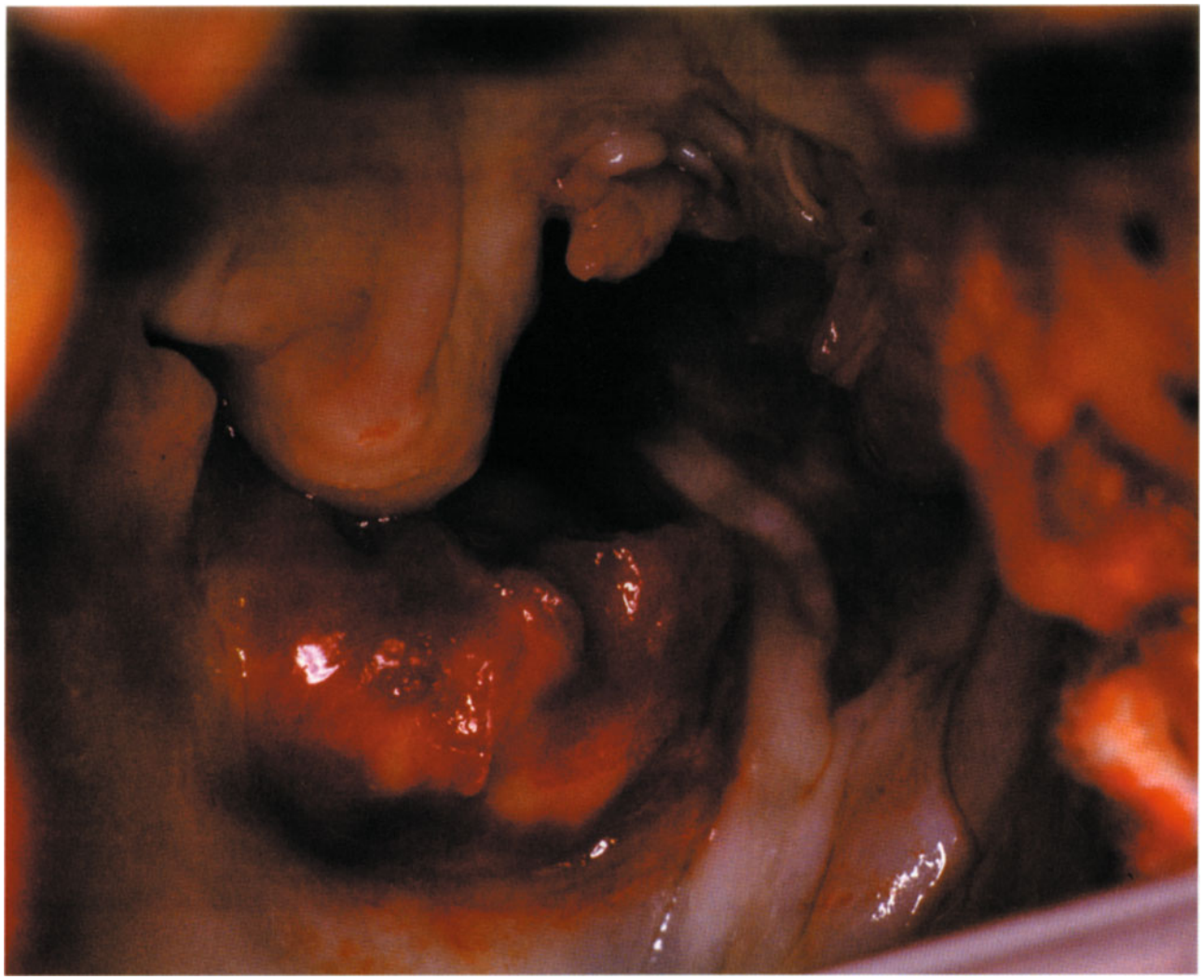


FIG. 16.4 (enlargement). Vegetations, damaged leaflets and annulus and extention of infection into the adjacent myocardium are seen

Mitral valve surgery for complications of endocarditis

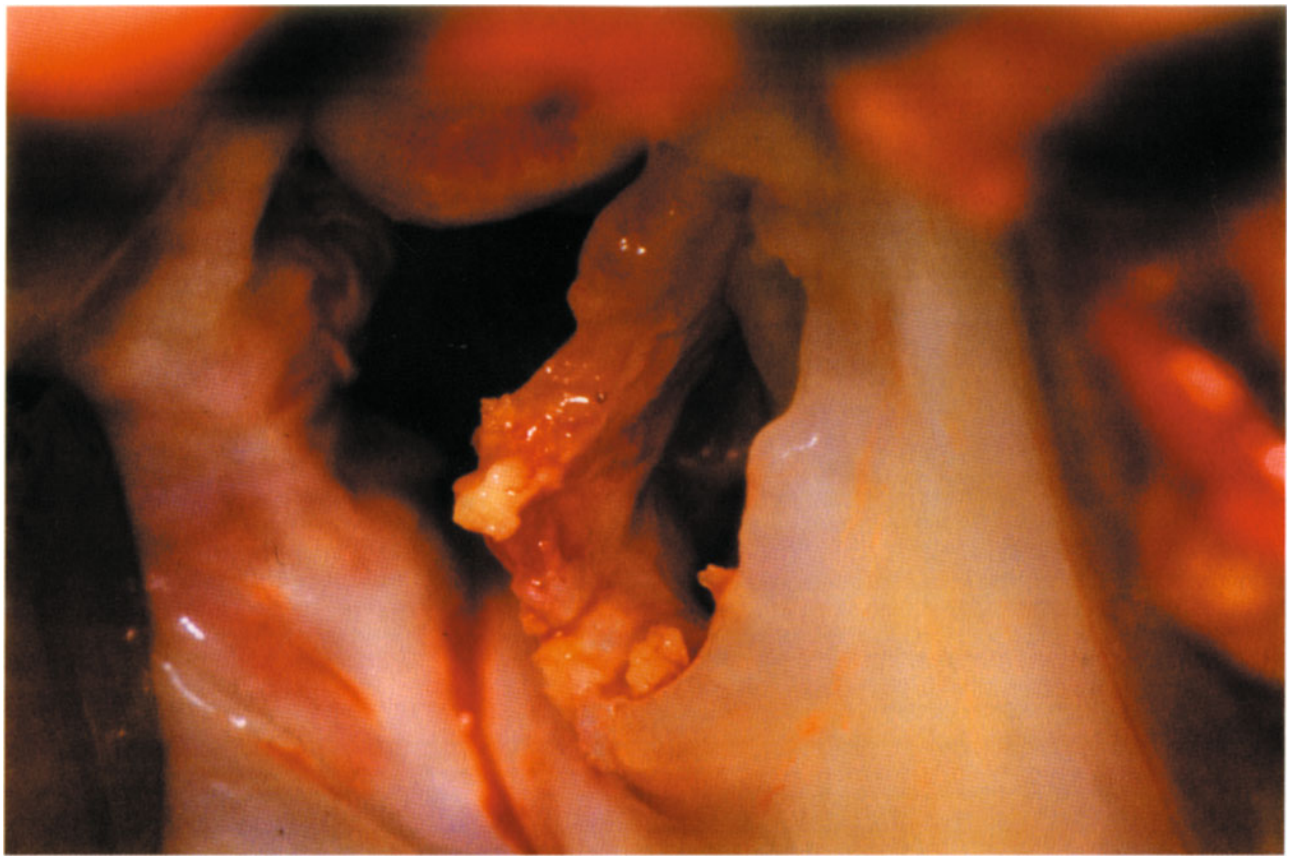


FIG. 19.2 (enlargement). TEE (longitudinal view) and intraoperative photography show a large area of endocarditis with posterior leaflet perforation. Involvement of the paracommissural segment of the anterior leaflet and the surrounding annulus with multiple vegetations is also demonstrated

Mitral valve surgery in the presence of active, infective aortic valve endocarditis

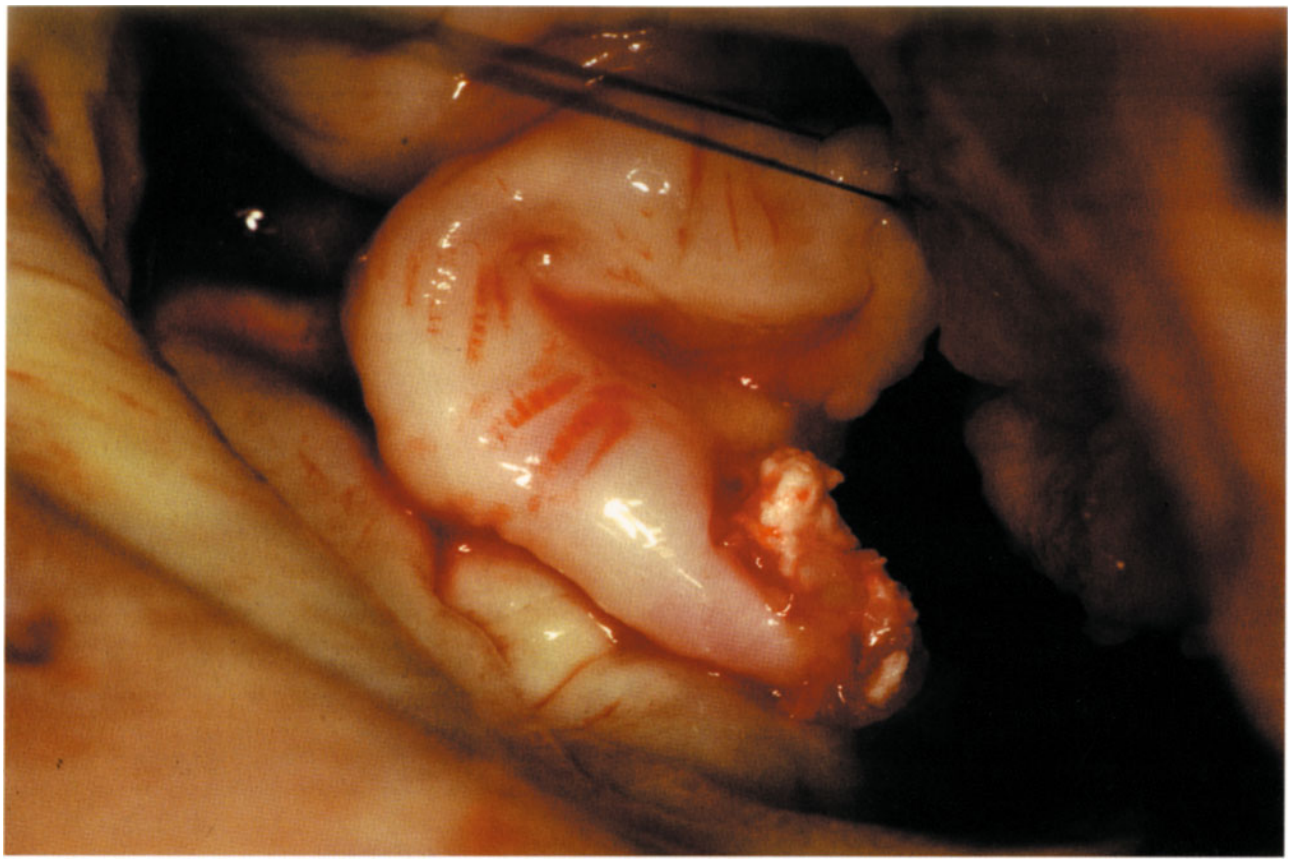


Fig. 20.4 (enlargement). Intraoperatively, cheese-like vegetations are noted in this patient with active fungal endocarditis.

7 Mitral valve surgery in the presence of leaflet restriction

7.1 Pathogenesis of rheumatic mitral valvular diseases

The primary effects of active rheumatic carditis are annular dilatation and an inflammatory valvulitis. Dilatation of atrioventricular and semilunar valve annuli is common. Dilatation of the mitral annulus may be extremely marked and measure as much as twice the normal area. Thus, significant mitral annulus dilatation does not occur with the ventricular dilatation associated with congestive cardiomyopathy, ischemic heart disease and longstanding aortic regurgitation. The rheumatic process damages collagen fibres and the ground substance of connective tissue in the heart. Concentration of acute inflammatory cells to the region of insertion of the posterior leaflet of the mitral valve into the basal left ventricular myocardium has also been known. The failing diffusely diseased myocardium affected by acute rheumatic carditis has a tendency to dilate. This tendency is not resisted by the weakened connective tissue of the mitral and aortic valve rings. Secondary changes supervene following mitral annular dilatation. When the mitral annulus dilates, the supportive keystone mechanism is lost and the angle between the aortic and mitral annuli is increased so that their relationship approximates the horizontal. The mitral leaflets and hence the chordopapillary mechanism are thus subjected to greater pressure and are under more tension during left ventricular ejection. Moreover, left ventricular dilatation also causes an inefficient lateral, rather than vertical, pull of the papillary muscles. All these factors contribute to increased tension on the chordae tendineae and leaflets, themselves weakened by the rheumatic process, and result in elongated or ruptured chordae tendineae and flail leaflets. Another consequence of mitral annular dilatation is the stretching of the posterior leaflet so that it may become functionally retracted. This process in turn may be further aggravated by left atrial dilatation and by true retraction of the posterior leaflet owing to its fibrosis or thickened, shortened chordae tendineae. With secondary involvement of other structures, valvulitis may also result in considerable scarring of the valve with commissural fusion, thickened leaflets, and fused chordae tendineae. These features are of a more chronic nature and account for the relative rarity of isolated mitral stenosis in the very young.

Preservation of the native mitral valve has obvious advantages, especially in children, young adults and patients in whom tightly controlled anticoagulation is rarely practicable and valve failure may be catastrophic.

Acute mitral regurgitation by rheumatic fever may require emergent valve surgery. The decision to operate urgently in these patients is based primarily on the degree of hemodynamic instability, not on the activity of the rheumatic fever. Mitral regurgitation in these cases may be due to rheumatic myocarditis affecting the papillary muscle. In some cases, however, global ventricular function as measured by echocardiography may be normal or increased. Further, combined aortic regurgitation and mitral regurgitation has been observed in our patients in acute rheumatic fever with normal ventricular function, suggesting that primary valvular involvement may also occur. Patients may develop mid to late systolic murmurs with mid-systolic clicks suggestive of mitral valve prolapse during acute rheumatic carditis. Angiography and echocardiography have observed transient systolic prolapse of the mitral valve in these cases, with eventual disappearance of the prolapse in the majority cases. Rheumatic mitral stenosis usually does not present as an acute valvular process, but patients may present emergently with pulmonary edema when a secondary condition such as fever or atrial fibrillation occurs. Massive and life threatening bronchial hemorrhage can also occur with rheumatic

mitral stenosis. This condition requires emergent surgical intervention.

7.2 Posterior leaflet extension patch plasty (Figs. 22, 29)

This method of valvuloplasty was applied in two groups of patients with type-III leaflet restriction (according to the classification of leaflet mobility).

Patients who, after rheumatic endocarditis, had severely fibrotic areas in the leaflets with tissue shrinkage, needed enlargement of the leaflet with a patch in order to bridge the coaptation gap between the two leaflets. The posterior leaflet was detached from the annulus from commissure to commissure. Calcified or diseased portions of the leaflet were resected. Subsequently, the fibrotic thickened chordae and papillary muscles were split longitudinally, to create free space for subvalvular mobility.

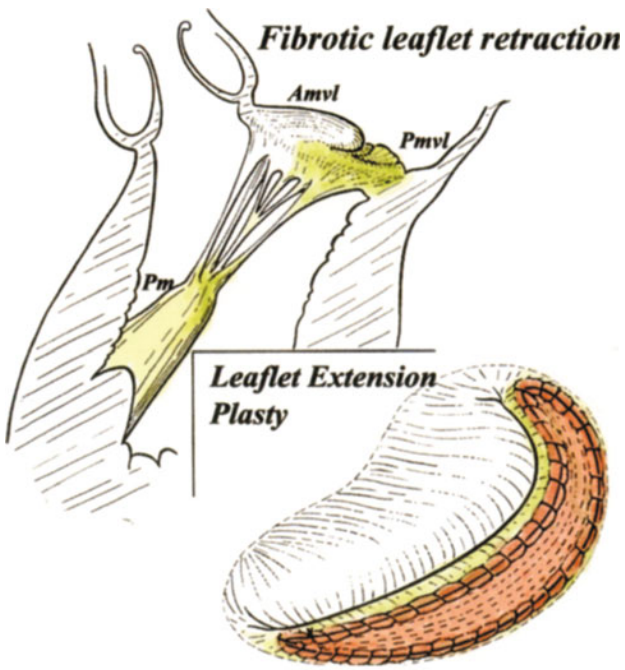


FIG. 22.1. Leaflet extension plasty with pericardium to treat the leaflet restriction and valvular fibrosis after chronic rheumatic carditis

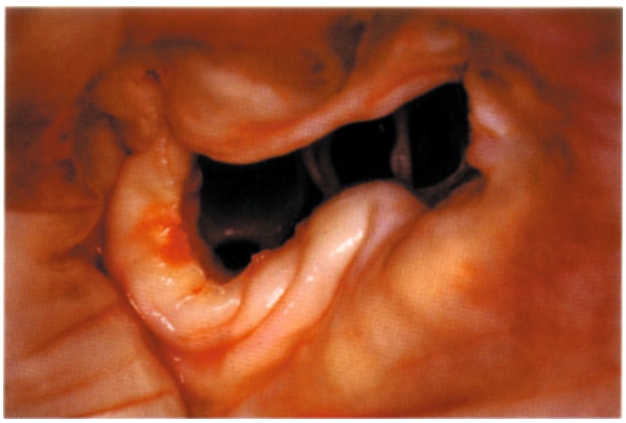


FIG. 22.2. Coaptation gap between the anterior and posterior leaflet due to shrinkage of the leaflet after rheumatic disease

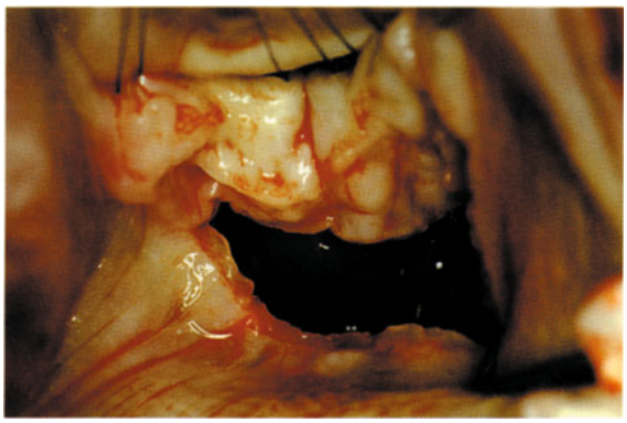


FIG. 22.3. The posterior leaflet is detached from commissure to commissure from the fibrotic annulus and is raised to the coaptation edge of the anterior leaflet with two 5-0 Prolene sutures. The gap thus created is filled with a patch of pericardium

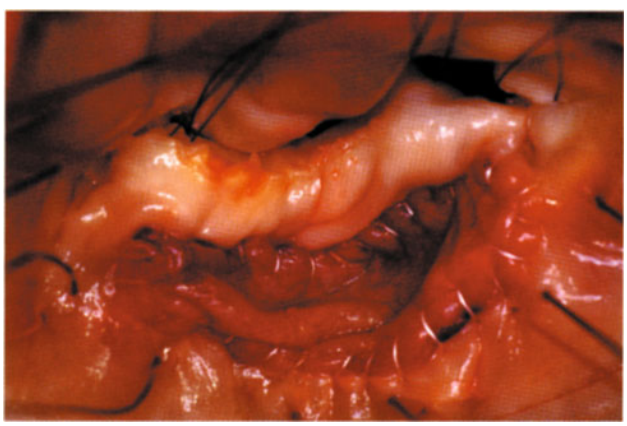


FIG. 22.4. An ovoid patch with dimensions ranging from 2.0 to 4.5 in length and 1.5 to 2.5 in width is adapted with 7-0 and 6-0 Gore Tex sutures and is used as leaflet replacement for leaflet extension plasty (up to more than 100%)

However, the chordae at the free edge were left untouched. An ovoid patch with dimensions ranging from 2.0 to 4.5 in length and 1.0 to 2.0 in width was then sutured to the annulus and to the mobilized posterior leaflet with a 6-0 or 7-0 PTFE continuous suture. The dimensions were calculated according to the width of the anterior leaflet with a 1:3 ratio in order not to hinder anterior movements during systole. The length of the patch was sized a few millimeters longer than the extent of the posterior leaflet incision. By doing so, the *surface could be extended by more than 100%* (Fig. 22.1–4).

A further group of patients with previous posterior wall infarction were also treated with this surgical method. The ischemic mitral regurgitation was the result of asymmetric posterior leaflet movement with left ventricular shifting after myocardial infarction. This group of patients with an ischemic cardiac pathology will be described in greater detail in the following pages (Fig. 29.1–8).

8 Combined reconstruction with leaflet extension plasty (Fig. 23)

This technique is especially used when patients with *combined defects*, whether of congenital or post-rheumatic origin, are subjected to reconstruction [36]. In addition to commissurotomy and mobilization of the leaflet by detaching adhesive and shortened chordae, fenestration of chorda bundles, or longitudinal splitting of the papillary muscle, the regurgitation component should also be eliminated by unilateral or bilateral leaflet extension plasty in order to achieve a sufficiently large orifice as well as complete closure of the valve (Fig. 23.1–8).

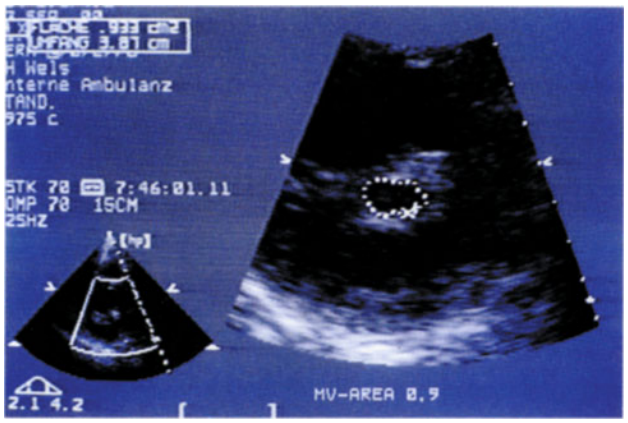


Fig. 23.1. The mitral valve orifice area (MVA) in a 35-year-old woman with rheumatic mitral stenosis with failed balloon dilatation measured 0.9 cm^2

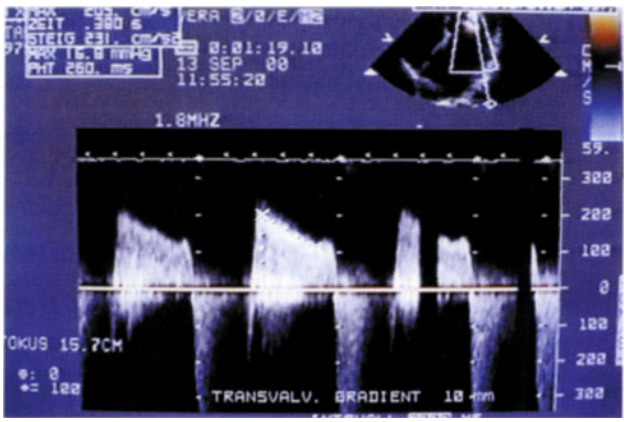


Fig. 23.2. Doppler recording discloses a transvalvular pressure gradient of 10 mm Hg and also mitral regurgitation

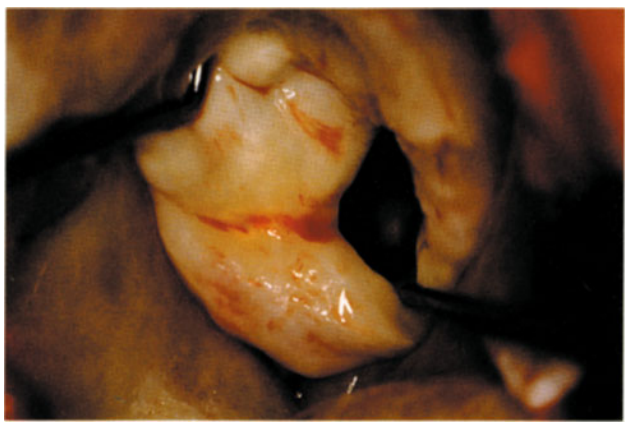


Fig. 23.3. The surgical analysis shows valvular stenosis and the mechanism for insufficiency. Primary congenital commissural fusion with subsequent rheumatic disease was causative for severe valvular fibrosis and shrinkage of leaflet tissue in this patient. All main chordae and the corresponding papillary muscle heads were extremely stiff and retracted, but not calcified

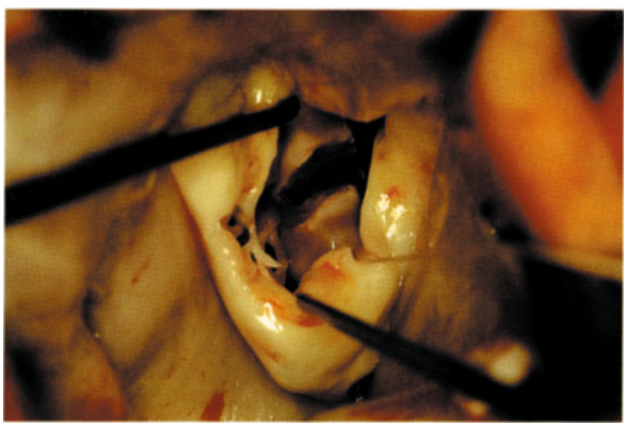


Fig. 23.4. First a commissurotomy up to both trigones was performed, then fenestration of the strong bundles of chordae and splitting of the papillary muscles were undertaken to achieve good mobility of the chordae

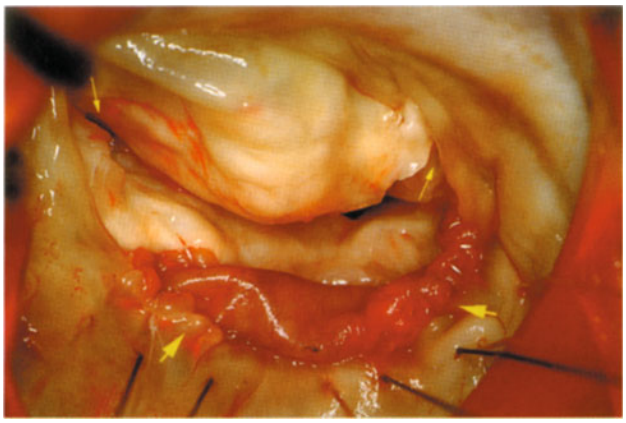
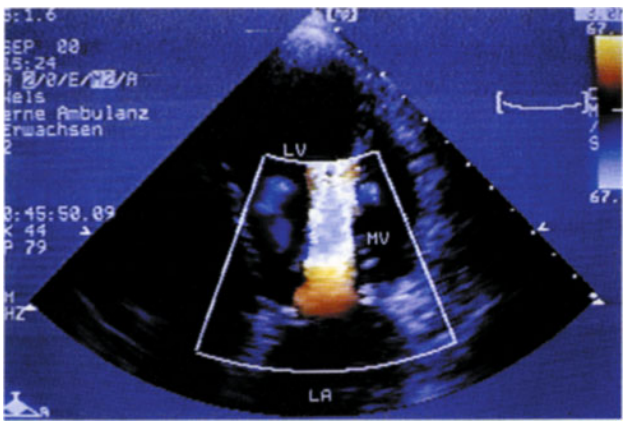


FIG. 23.5. Valvuloplasty with leaflet extension plasty was then performed. Only autologous pericardium was used as substitute for the shrunken, fibrotic tissue (lower arrow). This extension patch plasty permitted uninterrupted closure of the coapting edges of both leaflets. The upper arrow shows the extended mitral valve orifice after bilateral commissurotomy

An open mitral commissurotomy is generally more easily performed in non-calcified mitral stenoses which have developed adhesions at both commissures, and in which the body of the leaflet – especially that of the anterior leaflet which executes the actual closing mechanism – is still readily mobile and therefore able to achieve rapid mitral valve closure. Even thickening of the leaflet margins, also in the adhesive region of the commissures, does not hinder the achievement of better results after valvuloplasty with extension plasty than after mitral valve replacement (Fig. 23.5–8).



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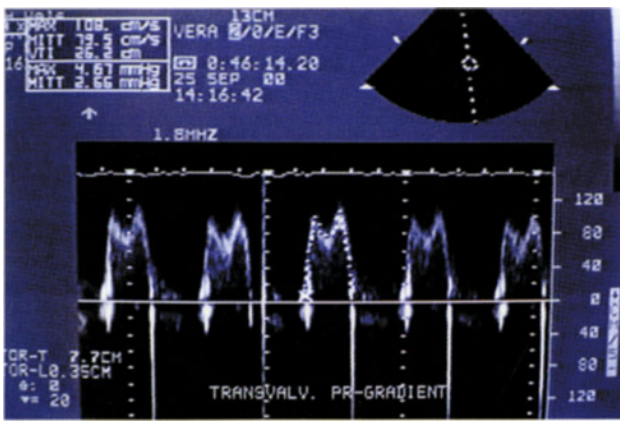

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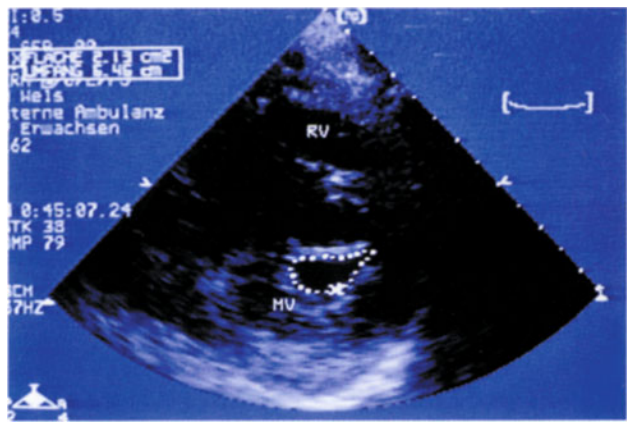


23.8

FIGS. 23.6–8. Show enlarged mitral valve orifice (2.1 cm^2), unimpeded flow, no mitral gradient and no mitral regurgitation

9 Mitral valve surgery in the presence of leaflet elongation

9.1 Chordal shortening (Fig. 24.1–3)

One option of valvuloplasty in cases of a prolapsed leaflet with elongation of the chordae is to correct the *length* of such chordae tendinae [16]. First, the extent of relative chorda elongation is measured by applying traction at both free edges of the leaflet with fine nerve hooks. Then, the body of the concerned papillary muscle is split longitudinally from the tip; the portion with elongated chordae is shifted and this is fixed to the rest of the muscle (Fig. 24.1–3).

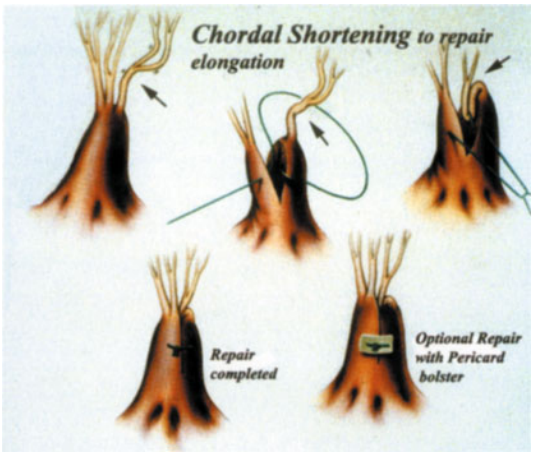


Fig. 24.2. Surgical illustration of chordae sliding plasty of the affected papillary muscle. The papillary muscle of the elongated chorda is split, the chorda is shifted to its detached counterpart and is sutured to it

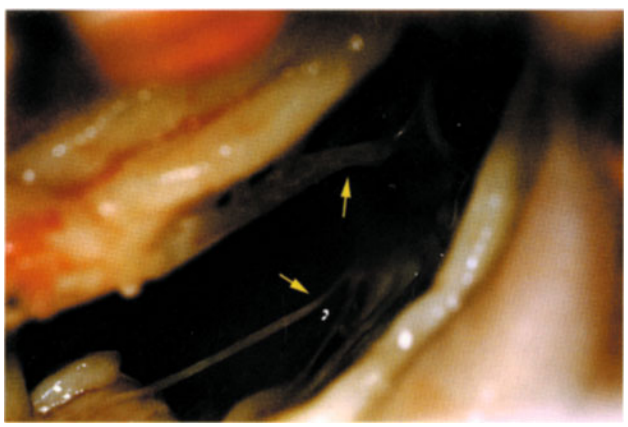


Fig. 24.1. Chordal elongation is one of the main causes of mitral valve prolapse (arrows)

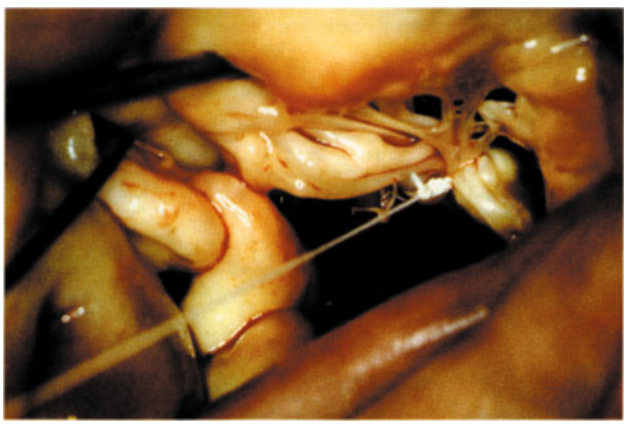


Fig. 24.3. Correction of a two-fold elongated anterior leaflet

9.2 Sliding plasty with chordal shortening (Fig. 24.4–6)

Sliding plasty with chordal shortening is often used not only in cases of prolapse of one leaflet but also *both leaflets* (Fig. 24.4). *In order to correct a complex bi-leaflet pathology, ideally, sliding plasty is used in combination with other surgical techniques.*

Figure 24.5 and 6 shows an example of leaflet sliding plasty for the treatment of a combined mitral defect with prolapse of the anterior leaflet and an infectious posterior leaflet. Using the combined technique in this case, not only is the prolapse corrected, but the infection is also successfully treated [29].

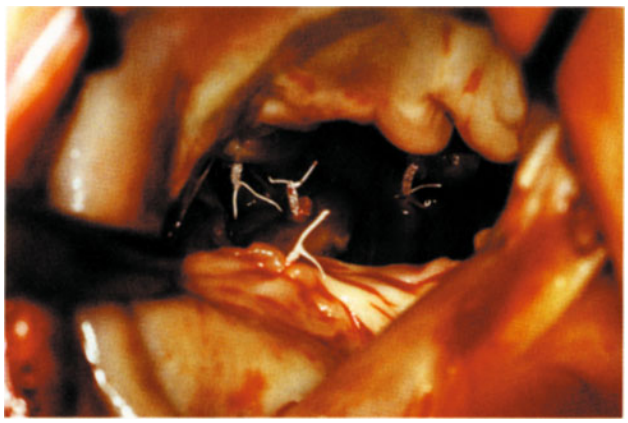


FIG. 24.4. Multiple chordal shortening towards the anterior and posterior leaflet

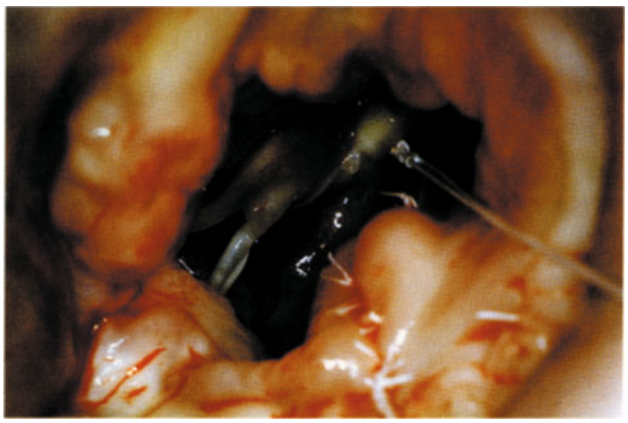


FIG. 24.5. Combination of leaflet sliding plasty to the posterior leaflet and chordal shortening at the anterior leaflet for the treatment of a complex valve pathology

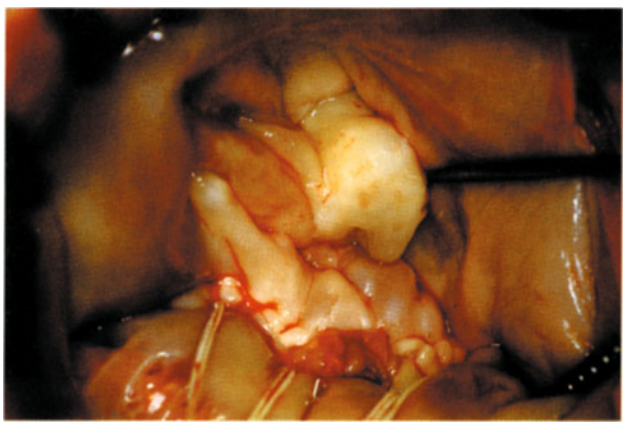


FIG. 24.6. Successful reconstruction combined with leaflet sliding plasty and chordal shortening (checked with nerve hooks). Excellent re-coaptation of both leaflets is seen without prolapse

10 Mitral valve surgery in the presence of flail leaflet

Ruptured chordae tendineae may involve chordae inserting into either leaflet. The causes are diverse and include infective endocarditis, acute rheumatic carditis, Marfan's syndrome or Barlow's disease, and direct or blunt cardiac trauma. In a large percentage of cases there is no identifiable cause, so-called idiopathic rupture of chordae tendineae. The syndrome of acute severe mitral regurgitation due to idiopathic chordal rupture should be readily recognized clinically. All cardiologists are familiar with the presentation of a patient with recent or sudden onset of severe breathlessness due to chordal rupture. The usual findings at surgery are that one or more chordae to the posterior leaflet or to the anterior leaflet or to both leaflets are ruptured. The leaflet itself is often ballooning as well as flail. The billowing may be localized to the middle scallop of the leaflet. Both leaflets may be voluminous and compatible with so-called floppy valve syndromes. The pathological examination concluded in the majority of flail leaflet that the mitral valve prolapse is the underlying morphologic abnormality.

Prolapse or chordal rupture of the anterior mitral leaflet has the reputation of being a difficult and problem, because of the poor results obtained after triangular anterior leaflet resection. Chordal transfer or replacements by Gore-Tex sutures are innovative surgical techniques in this respect, now readily repaired in the majority of cases, and with excellent results. Another alternative technique to treat this pathology is the application with a narrow strip of pretreated pericardial patch for chordal replacement.

Chordal transfer may be performed in two ways, namely with valve tissue (leaflet flip-over), and without valve tissue.

10.1 Chordal transposition plasty (Fig. 25)

Chordal rupture in the anterior leaflet, especially very severe rupture of the main chordae, whether after infectious or degenerative disease, can be successfully reconstructed by using valvuloplasty with the **leaflet flip-over** technique (Fig. 25A.1–4). **Leaflet flip-over** is a method of chordal transfer, whereby a small portion of the posterior leaflet opposite the ruptured chordae is detached together with primary or secondary chordae, whose continuity to the papillary muscle is kept intact [37]. This slice of leaflet is sutured to the underside of the anterior leaflet at insertion site of the ruptured chorda, using 7-0 or 8-0 PTFE sutures. The defect thus created in the posterior leaflet is closed by means of plication plasty.

Even strong **secondary chordae tendinae** from the anterior or posterior leaflet may be transposed to the defective segment by this procedure (Fig. 25B.1, 2). For chordae to be transferable without valve tissue, they must be of good quality, i.e. neither too thin, calcified nor altered by endocarditis.

An anteroposterior borderline divides the mitral valve into two areas corresponding to the supporting subvalvular apparatus: the chordae anchored on the posteromedian papillary muscle are directed to the "right" area, and on the anterolateral papillary muscle to the "left" area. Chordae transfer must be performed within the same area, in order to avoid borderline crossing that might result a restrictive motion.

However, prior to any planned flip-over technique, an exact preoperative echo investigation is mandatory in order to evaluate the feasibility of surgery. By doing so, the surgeon gains a more accurate impression of the prevailing conditions and does not need to face unpleasant surprises during surgery. Furthermore, the investigation permits timely detection of possible contraindications and thus allows the surgeon to consider a different surgical method in time [13].

Investigation with three dimensional echocardiography may provide us more detail information regarding the anatomical structure of the

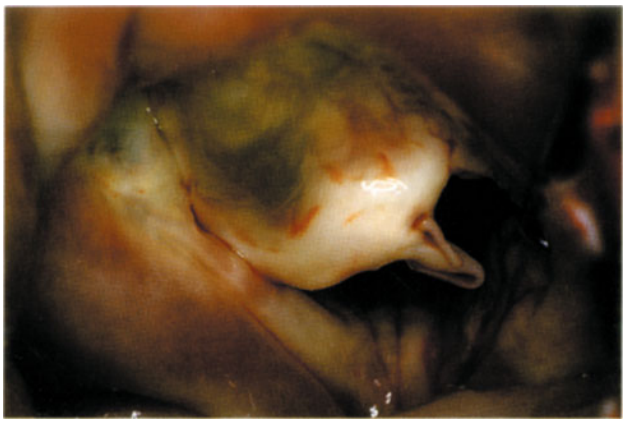


FIG. 25A.1. Rupture of the anterior leaflet chords

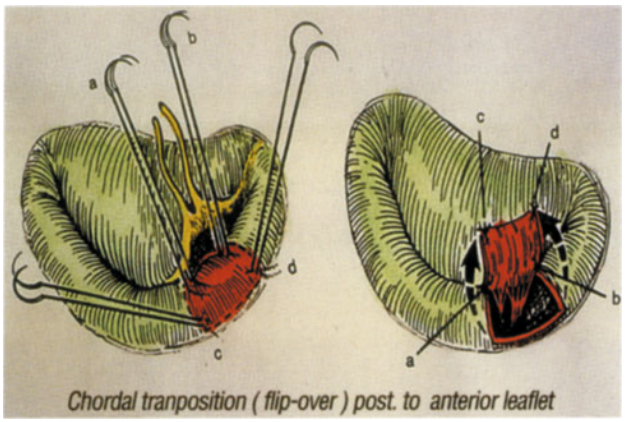


FIG. 25A.2. The illustration shows the surgical technique for preserving the anterior leaflet with a flip-over from the posterior to the anterior leaflet

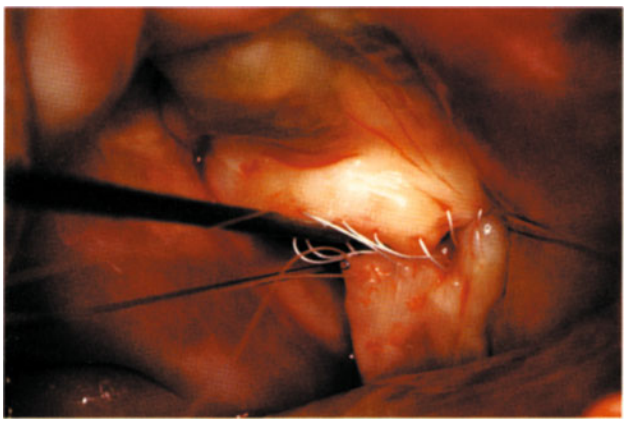


FIG. 25A.3. At surgery, fixation of the posterior leaflet to the coaptation edge of the anterior leaflet with 7-0 Gore Tex sutures

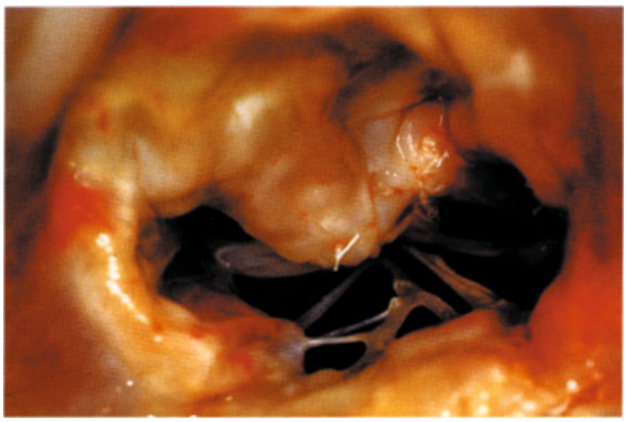


FIG. 25A.4. After successful transfer of the posterior chordae tendineae to the anterior leaflet

Secondary chordae transfer in mitral valve repair

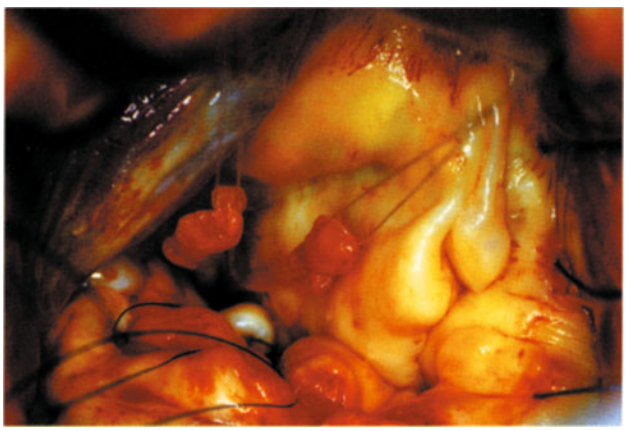


FIG. 25B.1. Two groups of secondary chordae tendineae from posterior leaflet are suspended with 7-0 PTFE sutures ready for implantation to anterior-lateral margin of anterior leaflet

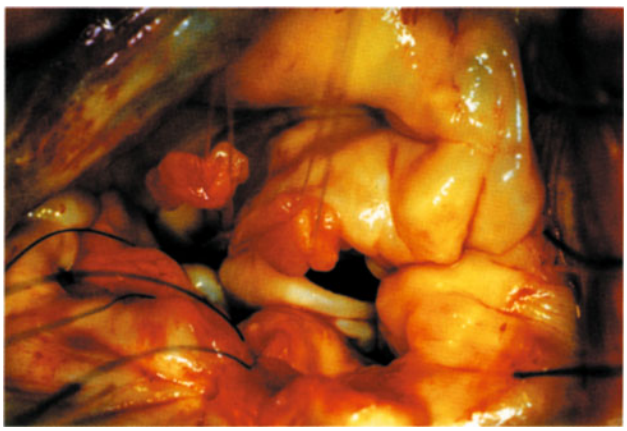


FIG. 25B.2. One group of the chordae tendineae has been attached to the coaptation edge of anterior leaflet

subvalvular apparatus prior the operation, before selection to use this repair technique.

10.2 Chordal replacement with a narrow strip of patch (Fig. 26)

The flip–over technique also has its limitations. The most important is noticed being prolapse of the segment of the posterior leaflet opposite that of the anterior leaflet. Another problem is the quality of the posterior leaflet tissue to be transferred. Abnormal, rigid posterior leaflet tissue transferred to the anterior leaflet can interfere substantially with its mobility, and the repair will fail.

When suitable chordae for transfer are not available in patients after aggressive tissue debridement, or when the patient has an additional pathology of the posterior leaflet (e.g., annulus calcification) (Fig. 26.1), then valve repair with chordal replacement using a narrow strip of patch is a meaningful procedure [38].

A strip of pre-treated pericardium about 4 mm wide and 4.5 cm long is tailored for chordal replacement. One end of the strip is sutured to the appropriate portion of the papillary muscle head with two to three 7-0 PTFE U sutures. The length of the "new chorda" is determined by comparison with the normal leaflet on the opposite side or with the commissural reference point (using a nerve hook) and was marked with a 7-0 suture under traction. The strip of pericardium is fixed to the ventricular surface of the prolapsed free edge, at the level of the marking, with two 7-0 PTFE sutures. Thus, exact apposition during leaflet closure is achieved (Fig. 26.3, 4).

Figure 26.1 and 2 shows the advantage of this method after a thorough preoperative echo study in a 74-year-old female patient. The patient had a flail leaflet pathology of the anterior leaflet, complicated by severe mitral valve insufficiency and reduced left ventricular function, but without atrial fibrillation. By way of *combined pathology*, the long-axis TEE revealed a *posterior annulus calcification* with normal leaflet mobility. A flip-over technique is not ideal in such

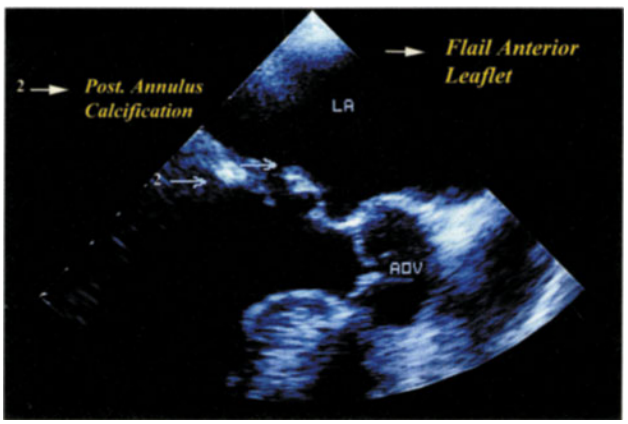


FIG. 26.1. The upper arrow on the long-axis TEE shows a flail anterior leaflet, the lower arrow shows the posterior annulus calcification in a 74-year-old woman with reduced left ventricular function without atrial fibrillation

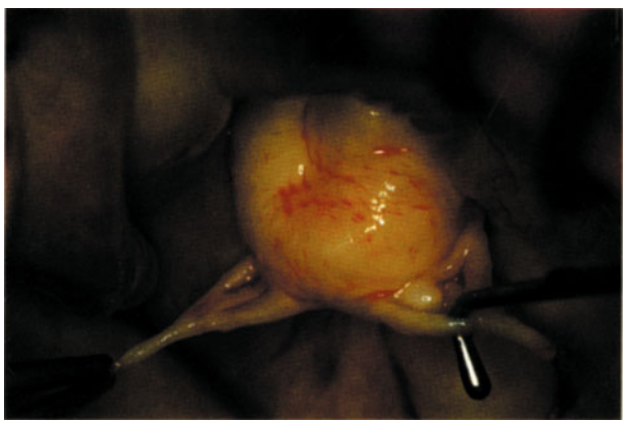


FIG. 26.2. Surgical photograph of the same patient with multiple rupture of multiple main chordae. As the preoperative TEE showed signs of posterior annulus calcification, the flip-over technique was not ideal in this case. A narrow autologous pericardial strip was used to replace the anterior chordae tendineae. When this is done, an additional surgical procedure in the posterior leaflet or annulus is not required

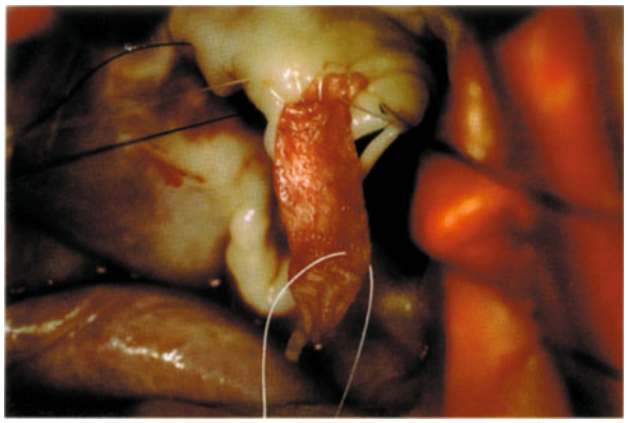


FIG. 26.3. A strip of glutaraldehyde-treated autologous pericardium is sutured to the coaptation edge of the anterior leaflet with 7-0 Gore Tex

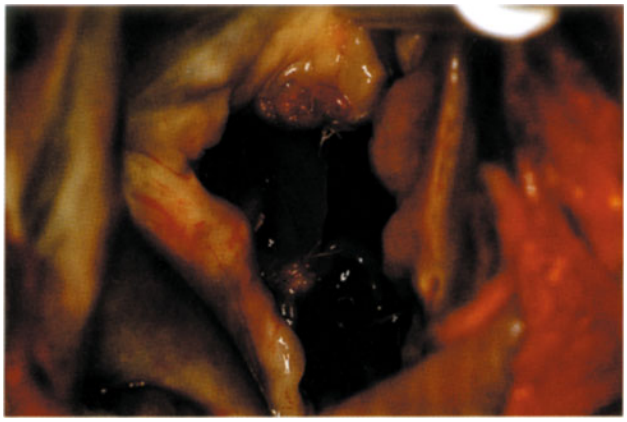


FIG. 26.4. Chordal replacement with autologous pericardium anchored to trabecular with 7-0 Gore Tex

cases, as the prolonged ischemic bypass time for correction of both pathological components markedly increases the surgical risk in an aged woman with restricted left ventricular function [9]. Valvuloplasty with a strip of pericardium is time-saving and more simple. The patient did not need to undergo an additional surgical procedure in the posterior leaflet and annulus. Having achieved a sinus rhythm, she really did benefit from the repair [3]–[5], [9].

Chordal transposition plasty

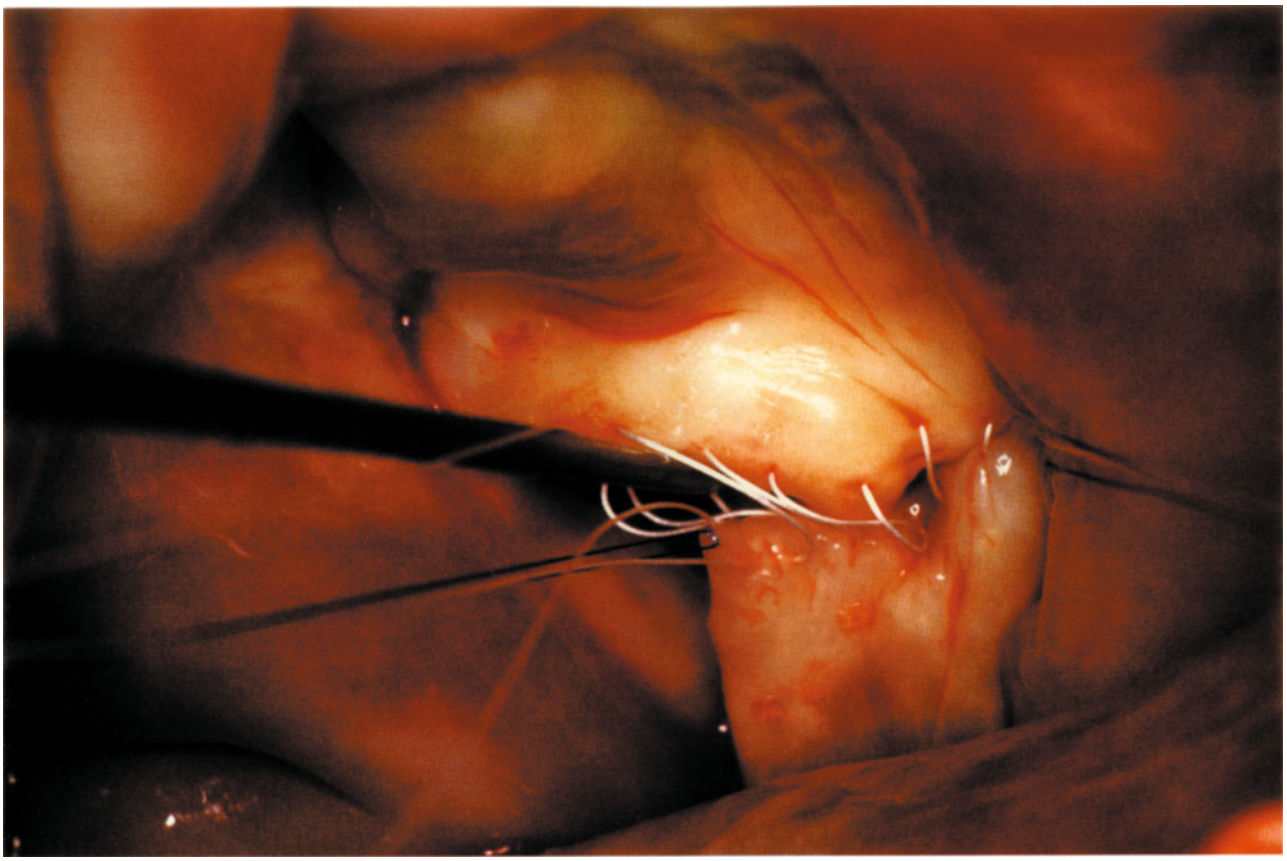


FIG. 25A.3 (enlargement). Flip-over technique. At surgery, fixation of the posterior leaflet to the coaptation edge of the anterior leaflet with 7-0 Gore Tex sutures

Mitral valve surgery in the presence of flail leaflet

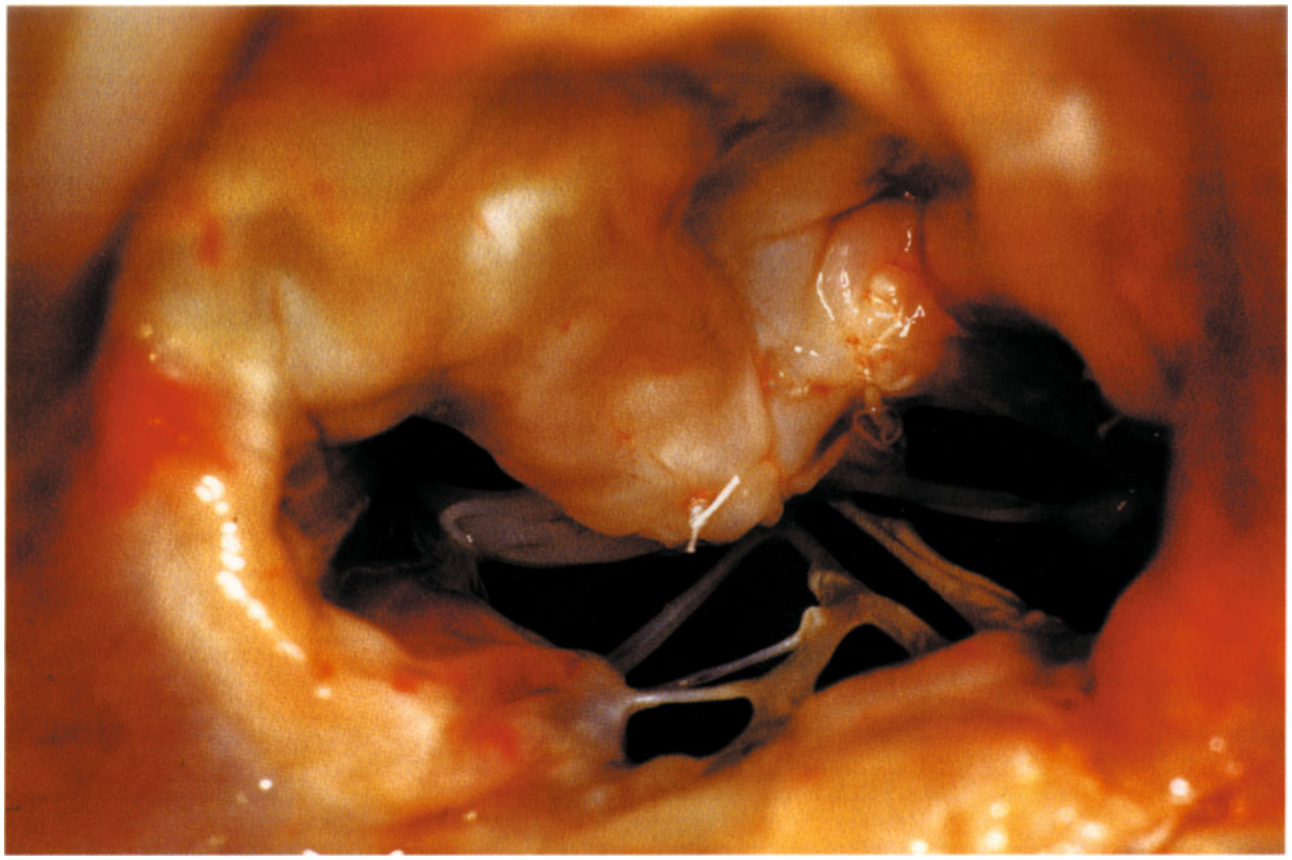


FIG. 25A.4 (enlargement).After successful transfer of the posterior chordae tendineae to the anterior leaflet

11 SURGERY FOR MITRAL APPARATUS DISORDERS CAUSED BY ISCHEMIC HEART DISEASE (Figs. 27–29)

Ischemic mitral valve disease ranks third among the most common causes of mitral insufficiency in surgical patients. Mitral insufficiency in association with coronary artery disease is well described and carries a worse prognosis with a higher operative risk compared with either rheumatic or degenerative etiologies. The incidence of concomitant mitral regurgitation in patients with coronary artery disease is reported to range between 6 and 19% worldwide! The reason usually is previous ischemic damage to the left ventricle.

Ischemic mitral regurgitation remains one of the most changing management problems in cardiac surgery. In our hospital, ischemia is the cause of mitral insufficiency in 6% of patients, the majority of patients are treated medically, but patients with severe regurgitation are found to benefit from surgical treatment. In the last decade, the operative approach to moderate or severe ischemic regurgitation has evolved from myocardial revascularization to surgical revascularization combined with valvular intervention.

Previous surgical management of ischemic mitral regurgitation with prosthetic valve replacement has been suboptimal. In the past studies of our patients with chronic regurgitation, 35% were found prolapse and 65% restricted leaflet motion. Left ventricular function was significantly worse in our patients with restricted leaflet motion. Restricted leaflet motion was associated with higher risk for late death in patients with both acute and chronic mitral regurgitation has been known. Studies compiled the large series of cases of mitral valve repair for ischemic mitral regurgitation have demonstrated that left ventricular function is the most determinant of both early and long-term result. The global left ventricular dysfunction contributes to recurrent mitral regurgitation post-repair. The fact that recent preoperative myocardial infarction, but not postoperative segmental wall motion abnormality, is associated with recurrent mitral regurgitation. Cardiac-related late deaths frequently occurred in patients with restricted leaflet motion in whom ventricular function was most impaired. In order to prevent further damage to the left ventricular pump function, these ischemic patients especially should be subjected to valve-conserving surgery as far as possible. A lower operative mortality with valvular repair for ischemic disease has been previously documented. When compared with previously published data, early survival benefit of valve reconstruction for ischemic insufficiency has previously been most marked in patients with recent myocardial infarction [39]–[41].

Prior to operation, the diagnosis of ischemic mitral regurgitation can be challenging. Angiography has technical limitations and cannot define anatomical – and therefore etiological considerations. Echocardiography with quantitative Doppler methods is essential. Because prognosis with ischemic mitral regurgitation has been associated with the degree of insufficiency, accurate quantitation of the condition is very important. Stress echocardiography is valued for patients suspected of having ischemic mitral regurgitation clinically when the resting echocardiography identifies mild mitral insufficiency. We recommend performing intraoperative echocardiography dynamic testing with volume stressing or pressure provocation with patients anaesthetized on a regular basis if the diagnosis is unclear, or if the degree of regurgitation is moderate, in the presence of intermittent or grade 2+ ischemic mitral regurgitation.

11.1 Intraoperative dynamic testing

A Swan-Ganz catheter is inserted, a midline sternotomy is performed and the aorta is cannu-

lated. The degree and the mechanism of mitral regurgitation are now assessed by mean of transesophageal echocardiography. Under anesthesia, it is not surprising to observe a decrease of mitral regurgitation when compared with preoperative transesophageal echocardiography. A volume-loading test is then performed: the pulmonary capillary wedge pressure is raised by at least 10–15 mmHg by a rapid filling via the aortic cannula. If the ischemic mitral regurgitation does not significantly increase, the manoeuvre is completed by a pressure-afterload test: a 5.0 mg bolus of ephedrine is given intravenously. Ephedrine acts exclusively on the systemic peripheral resistance and has no inotropic effect. A mitral valve surgery is only decided if the dynamic testing is positive, that is if it results in a definite worsening of the ischemic mitral regurgitation.

The routine use of transesophageal echocardiography assists to identify valve prolapse results from papillary muscle infarction with elongation, paresis of the papillary muscle, or papillary muscle rupture, restriction of leaflet motion occurs with regional or global ventricular dilation. Recognizing the functional basis for regurgitation in these valves not only dictates the surgical approach, but also provides for a useful predictor of long-term result. Occasionally, complex valve repair involving with concomitant coronary artery bypass or multivalvular procedure is not appropriate because of associated valve pathology or other major leaflet abnormalities that are more suitably treated with prosthetic valve replacement. Caution should be applied in the case of patients with acute insufficiency related to very poor general condition requiring a short and definite procedure, not to prolong unduly an already lengthy aorta cross-clamp time. Currently, the majority of patients in our hospital with ischemic regurgitation are amenable to reconstructive techniques describing here.

11.2 Reimplantation of a ruptured head of the papillary muscle

Ischemic rupture of the papillary muscle is an urgent indication for surgery. Without immediate surgical treatment, patients who suffer complete rupture of the papillary muscle head would die of acute heart failure (Fig. 27.1) [42].

Once a papillary muscle rupture is confirmed by echo investigation, the patient must be operated on immediately. A rupture of a papillary muscle head is visualized in the TEE views as a floating muscle mass either in the left atrium (Fig. 27.2, 3) or in the left ventricle (Fig. 28.1, 2).

Reimplantation of a ruptured papillary muscle head in the trabecular network of the left ventricle is a promising approach. However, only very delicate suture material such as 7-0 PTFE should be used in conjunction with an autologous pericardial patch (Fig. 28.3, 4). Gross suture material may injure the trabecular network of the left ventricle and may cause disruption of the muscle even during knotting.

Severe ischemic mitral insufficiency due to papillary muscle rupture is an acute illness, and the physician's diagnostic and therapeutic decisions must be accomplished rapidly. Necrosis of papillary muscle will occur if surgery is delayed for months (Fig. 28B). This will make papillary muscle reattachment to the trabecular of the left ventricle, which is elegantly simple impossible. Moreover, the reparative technique will be more difficult, in which in such situation not unusual, patients are often precluded from advantageous repair with prosthetic replacement as alternative solution.

11.3 Posterior leaflet extension patch plasty for ischemic disease (Fig. 29)

After posterior wall infarction, a local contraction abnormality of a papillary muscle and the adjoining myocardial segment (preferably pos-

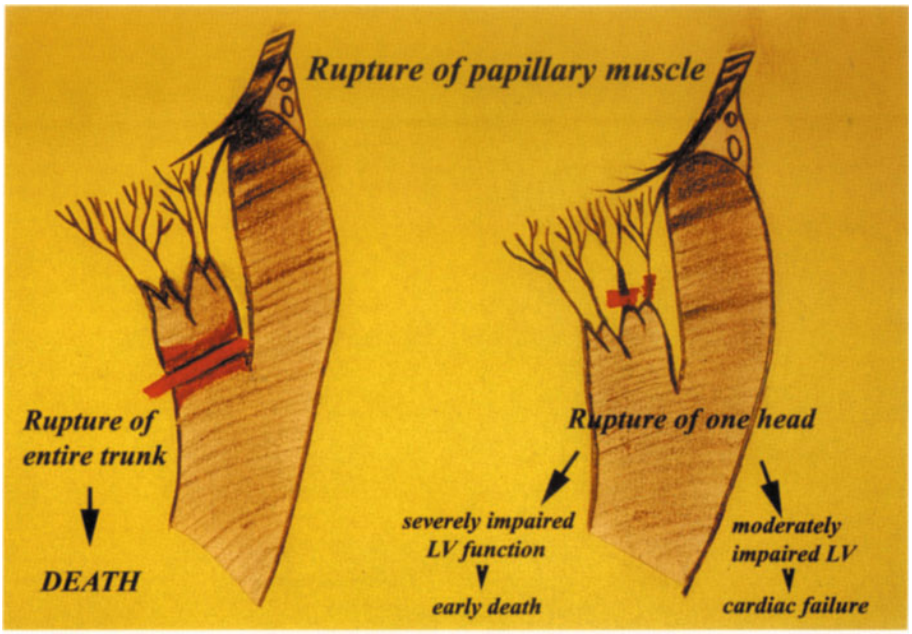


FIG. 27.1. Ischemic papillary muscle rupture is an urgent indication for surgery because acute severe mitral regurgitation usually results in cardiogenic shock and death

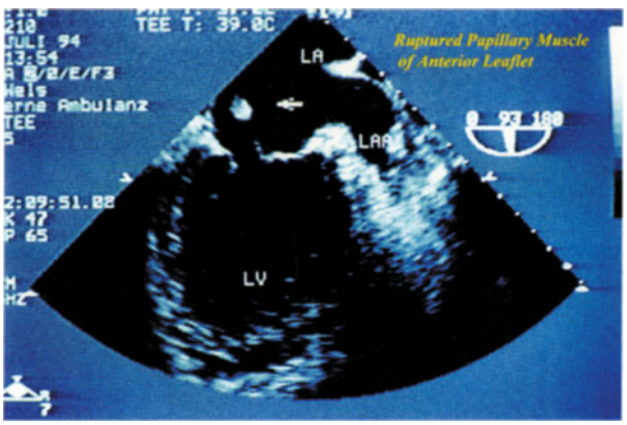


FIG. 27.2. Ruptured papillary muscle head of the anterior leaflet is visualized in the left atrium on the TEE two-chamber view

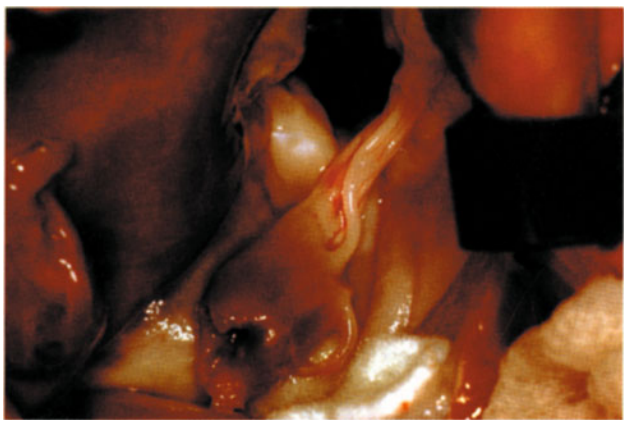


FIG. 27.3. Ruptured papillary muscle head in the left atrium prior to reconstruction

terolateral) is the pathophysiologic mechanism that frequently causes a functional disorder of the mitral valve [43]–[45]. On echocardiography, it is characterized by a coaptation defect with restriction of leaflet mobility (type III according to the classification of leaflet mobility).

In the apical two-chamber view investigation, one finds the posterolateral scar with the infarcted papillary muscle on the one hand, and the coaptation defect between the anterior and the contracted posterior mitral leaflet on the other. One observes a severe excentric mitral valve regurgitation extending up to the roof of the atrium (Fig. 29.1, 2).

Any degree of mitral regurgitation in an infarcted ventricle initiates left ventricular

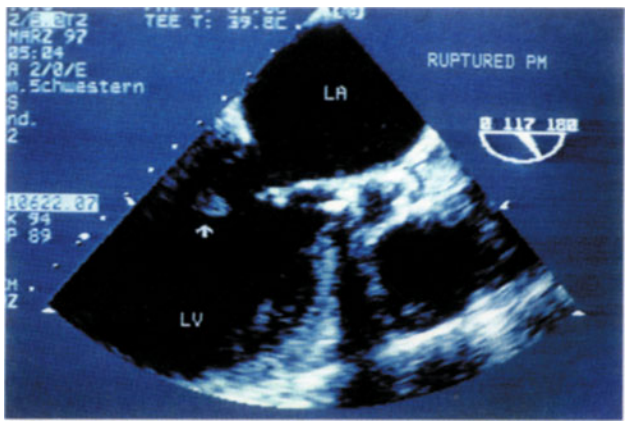


FIG. 28.1. Rupture of the papillary muscle trunk (modified TEE long-axis view) is an indication for acute surgical treatment to avoid cardiac death

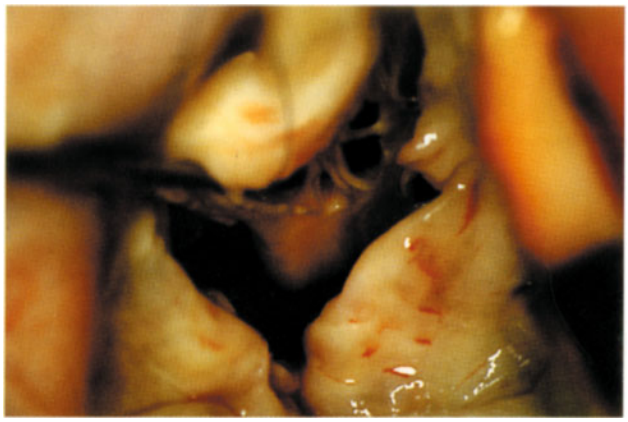


FIG. 28.2. Rupture of the papillary muscle trunk in the left ventricle, and numerous chordae tendineae hanging at the papillary muscle trunk are seen

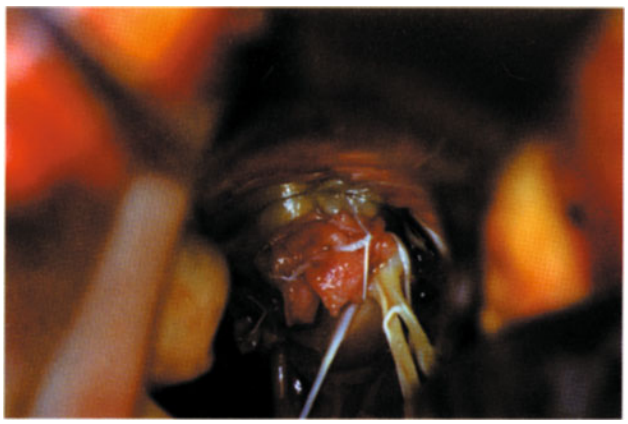


FIG. 28.3. Reimplantation of the ruptured papillary muscle trunk in the trabeculae of the left ventricle with 7-0 Gore Tex sutures, reinforced with an autologous pericardial patch

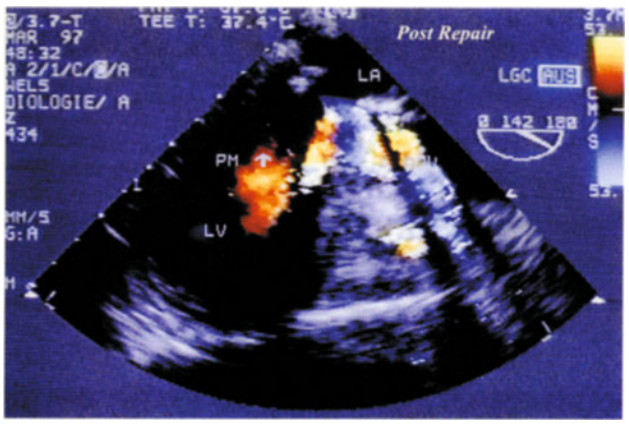


FIG. 28.4. The postoperative echocardiographic image shows successful valve repair without mitral insufficiency

remodeling changes that gradually lead to progressive left ventricular enlargement, loss of contractile force and more severe mitral regurgitation. The infarction of the papillary muscle and the surrounding myocardium causes shrinkage and shortening of the suspension apparatus of the mitral valve. This results in restricted mobility of the mitral leaflet and distorted geometry of mitral valve closure. The myocardial infarction may also lead to dilatation of the annulus and, in severe cases, to global dilatation of the left ventricle. Annulus dilatation, coaptation defects of the mitral leaflet and discoordination of the papillary muscles eventually lead to severe mitral insufficiency (Fig. 29.3, 4) [39], [40], [43], [46].

Ischemic mitral regurgitation is an extremely common complication of myocardial infarction and significantly reduces short- and long-term survival. The valve is structurally normal; it needs not be replaced. In *uncomplicated cases* without massive left ventricular enlargement and in the presence of largely preserved left ventricular function, an annuloplasty ring can either completely or substantially rectify the mitral regurgitation. However, severe asymmetric displacement of the papillary muscles after infarction could not always be normalized with an

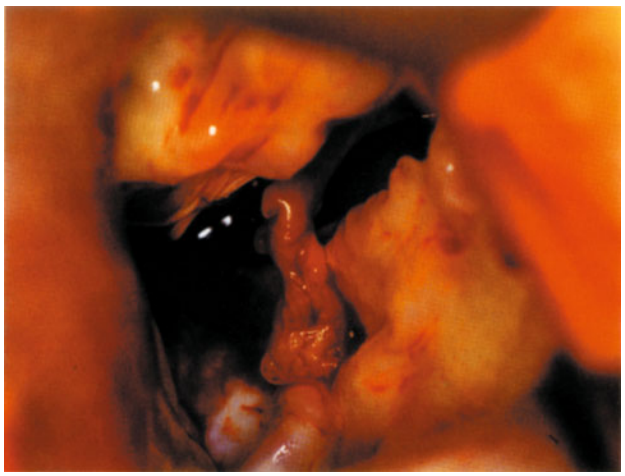


FIG. 28B. Severe necrosis of anterior papillary muscle occurred two and half months after myocardial infarction. This autolysis of muscle has made the reimplantation to the trabecular of the left ventricle impossible

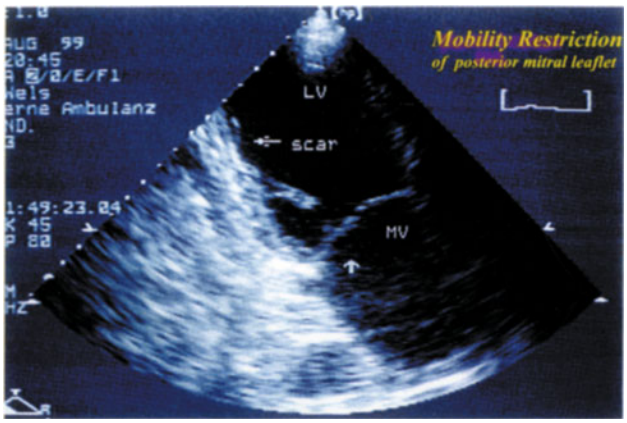


FIG. 29A.1. Apical two-chamber TTE view: The arrows show the posterolateral scar with the infarcted papillary muscle on the one hand, and the coaptation gap between the anterior and the retracted posterior mitral leaflet on the other

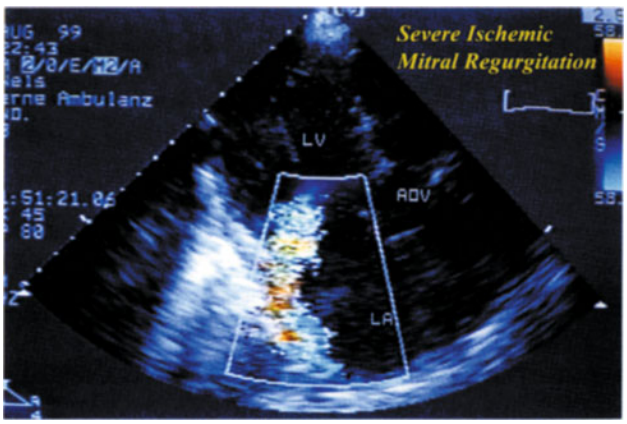


FIG. 29A.2. Color Doppler shows severe excentric mitral regurgitation

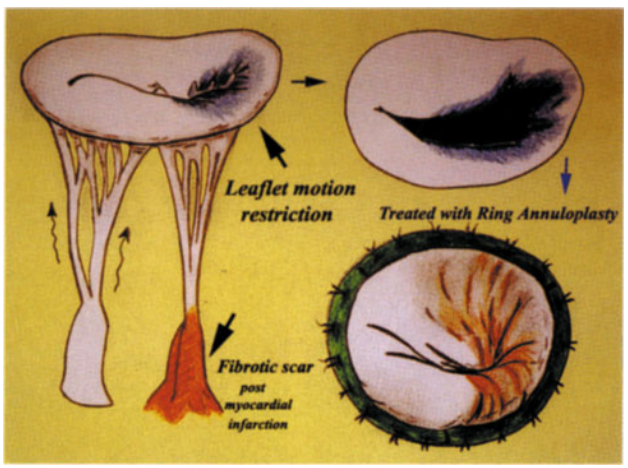
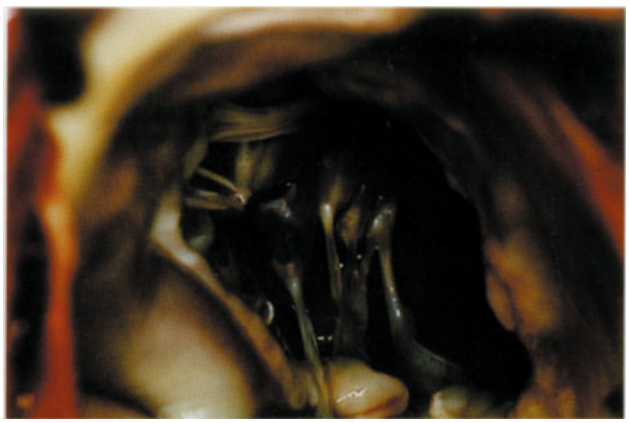


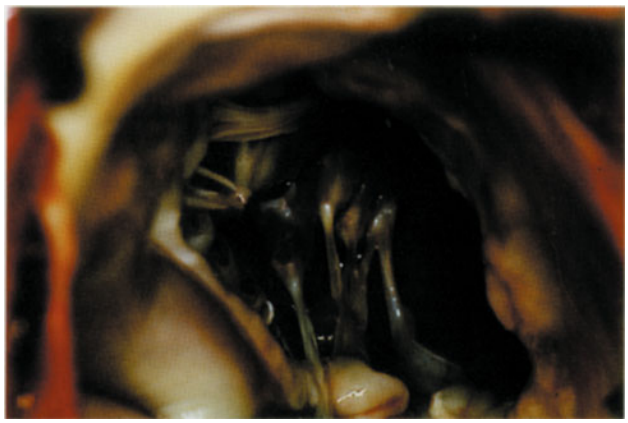
FIG. 29A.3. Infarction of the papillary muscle and the adjacent myocardium leads to shrinkage and shortening of the suspension apparatus of the mitral valve. This results in limited mobility of the mitral leaflet and incomplete mitral leaflet coaptation. Myocardial infarction may lead to annulus dilatation and, in severe cases, to global dilatation of the left ventricle. Annulus dilatation, coaptation gaps of the mitral leaflet and discoordination of the papillary muscles eventually lead to severe mitral insufficiency

annuloplasty ring alone. Single ring placement is often not efficacious and fails to eliminate severe mitral regurgitation in every patient and known with complication of valve stenosis [43], [44], [47], [48].

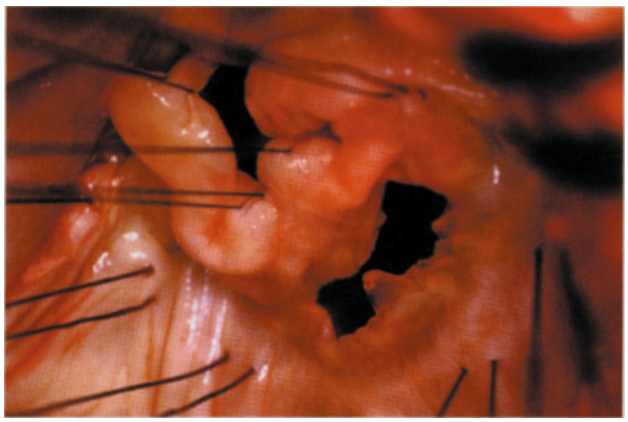
An innovative surgical procedure has been developed to ensure repair of the ischemic valve with restoration of valve mobility and the relationship between papillary muscles and the annulus after free mobilization of the fibrotic



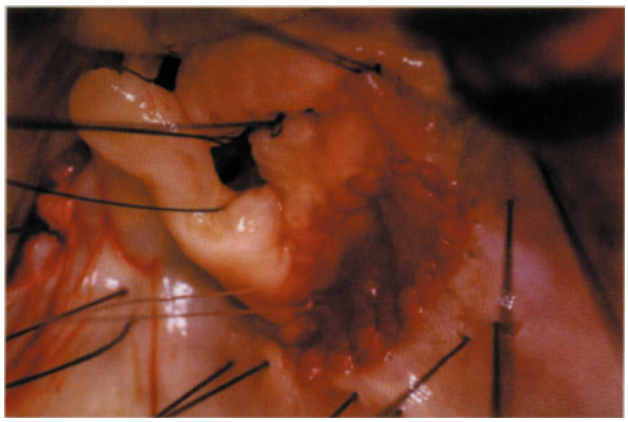
29A.4



29A.5



29A.6



29A.7

29A.8

FIGS. 29A.4–A.8. Valvuloplasty, with restoration of valve mobility and the relationship between papillary muscle and annulus after free mobilization of the fibrotic papillary muscle, is achieved after enlargement of the surface of the posterior mitral leaflet by means of leaflet extension plasty with autologous pericardium. The leaflet edges are approximated to each other so that coaptation is achieved. Anchoring of an annuloplasty ring is obligated to ensure correction of annulus dilatation and to facilitate improved coaptation of both leaflets so that mitral regurgitation

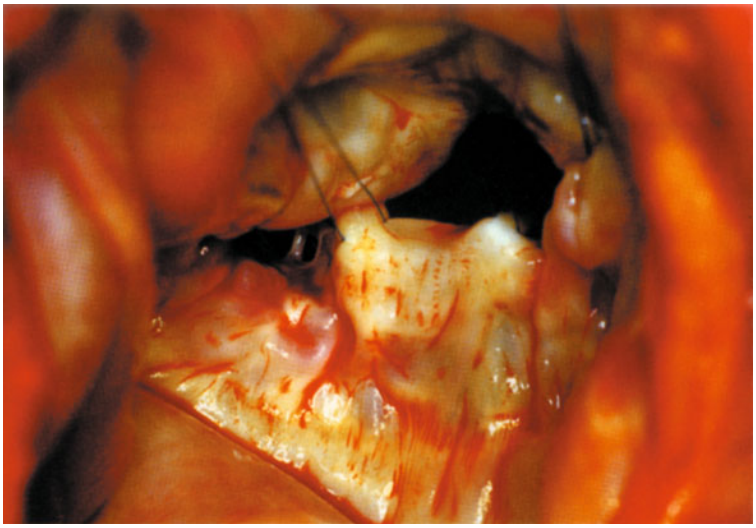


FIG. 29B.1. *This valve is structurally normal, it needs not be replaced.* The huge coaptation gap is due to posterior leaflet restriction. A dyskinetic/akinetic wall with apical displacement of the papillary muscle was found in preoperative echocardiographic diagnosis in this patient after a transmural posteromedial myocardial infarction that had occurred several years previously

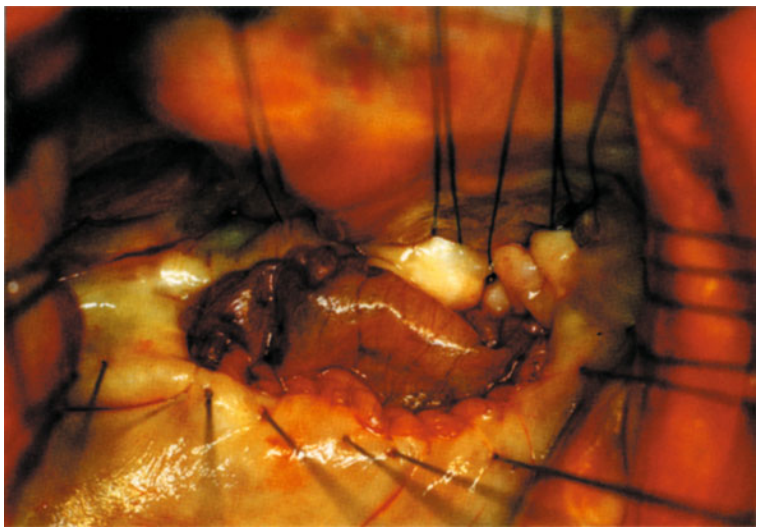


FIG. 29B.2. Patch enlargement of the restricted posterior leaflet demonstrated here has shown to provide an increase of more than 80% of the surface area. This remodeling technique allows restoring both the normal extent of mitral leaflet coaptation and posterior leaflet motion, which corrects the underlying asymmetric tethering of the papillary muscle to the myocardium. The coaptation gap shown in Fig. 29B.1 is now vanished with a new coaptation surface and abolished mitral regurgitation

papillary muscles is achieved after enlarging the surface area of the posterior mitral leaflet by means of leaflet extension plasty using autologous pericardium. The edges of the leaflet are approximated and, by doing so, coaptation is ensured (Fig. 29.5–8). This new surgical strategy in conjunction with an annuloplasty ring for reducing the annulus dilatation can successfully eliminate severe mitral insufficiency in patients with restricted leaflet mobility (Fig. 29B.1, 2) [23], [45], [49].

12 Operations in the atrioventricular valves in the presence of blunt cardiac trauma (Figs. 30–32)

Road accidents, sporting injuries and injuries at work are steadily increasing in our mechanized civilization. As the patients usually suffer multiple traumas, a blunt cardiac trauma is frequently overlooked at first and eventually seals the fate of the patient. It is the most common visceral injury associated with a fatal outcome. Blunt thoracic injury should always raise the suspicion of damage to the ribs, sternum, thoracic viscera, or spinal column. Serial ECG and enzyme measurement have been the historic method of diagnosis. These should raise suspicion, but specificity is low, hence echocardiography, transthoracic or transesophageal, is the investigation of choice, providing us a real time window for assessing myocardial injury and function. Echocardiography is inexpensive, rapid, and the procedure can be performed safely at the bedside. It is worth to realize the limits of TTE in the trauma patient due to tenderness and the presence of operative dressings; air interference from pneumothorax, mechanical ventilation, or mediastinal emphysema; an inability to position the patient, and not hesitate to use transesophageal approach. Intraoperative TEE can be performed in most preoperative procedures to assess pericardial diseases (i.e., fluid tamponade) and to evaluate ventricular function. The power of resolution of transesophageal sonography is superior to TTE for most intrathoracic pathologic processes, including papillary muscle rupture. Transesophageal approach should be considered as an adjunctive diagnostic modality in traumatic patients [50].

The mechanism behind mitral or tricuspid regurgitation following blunt cardiac trauma after rupture of the valve leaflet and papillary muscles or the chordae tendinae (Fig. 30.1) is known as the "*water hammer effect*". Sudden increase in pressure in the left and right ventricle leads to valve injury [51].

The most common injury after blunt chest trauma is myocardial contusion, which itself is probably underdiagnosed. Post-mortem series suggest that the right ventricle is most commonly involved, even though that acute valvular dysfunction is rare. Valvular injury may consist of papillary muscle rupture, chordae tendineae rupture, or laceration of the valve leaflet. Papillary muscle rupture may occur as a complete or partial event. Each papillary muscle is supplied by a long central artery arising from an epicardial vessel, and, if damaged, the muscle tip may undergo progressive ischemia and subsequent infarction, resulting in the delayed rupture.

Blunt injury to the cardiac valves lead to progressive ventricular failure often requiring immediately surgical management. With increasing experience, preservation of autogenous tissue to repair traumatic valve lesions is desirable and achievable, and has potential advantages over replacement. This should encourage consideration of repair rather than automatic valve replacement.

An unusual case of traumatic rupture of anterior and posterior papillary muscles and its management is presented first. This will follow with other cases of leaflet laceration and chordae rupture:

12.1 Early reconstruction

Simultaneous rupture of right ventricular papillary muscle heads towards the anterior and posterior leaflet inevitably has serious hemodynamic effects (Fig. 30.2). This condition cannot be tolerated for very long. Early surgical treatment with reimplantation of the papillary muscles is mandatory [52], [53]. Any delay in surgery promotes atrophy of the papillary muscles secondary to autolysis/necrosis. The muscle retraction increases in direct proportion to surgical inactivity. At a later point in time,

when the papillary muscles have shrunk, reimplantation of papillary muscles can no longer be performed. The goal should be *early reconstruction* to preserve the valve [51], [53], [54]. By using autologous pericardial patches, both papillary muscles are reimplanted in the adjacent trabeculae with 7-0 PTFE sutures (Fig. 30.3). Uninterrupted re-coaptation of the three leaflets, functioning in harmony, must subsequently be confirmed on TEE (Fig. 30.4).

Nearly one half of the anterior leaflet was detached in a 68-year-old woman (Fig. 31.1–3). This was not due to a penetrating injury to the heart but the outcome of leaflet rupture with blunt cardiac trauma after a *road accident* (so-called steering wheel syndrome). This condition

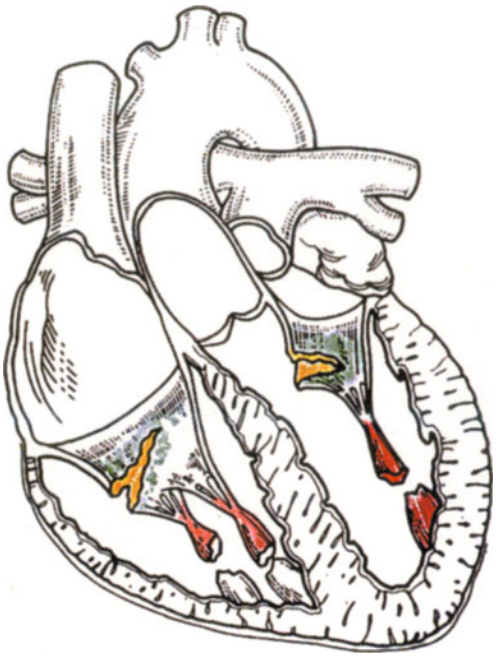


FIG. 30.1. Blunt cardiac trauma with rupture of valve leaflets and papillary muscles or chordae tendineae by the so-called "water hammer effect" can result in mitral insufficiency and/or tricuspid insufficiency. Trauma repair is usually by means of papillary muscle reimplantation

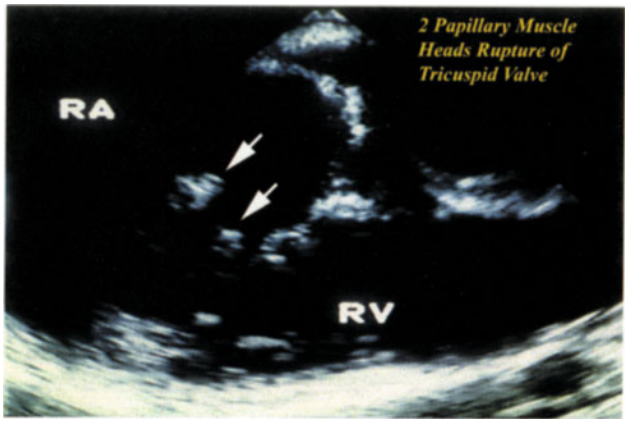


FIG. 30.2. Rupture of two papillary muscle heads of the tricuspid valve on the TEE of a 17 year-old man after a motorcycle accident

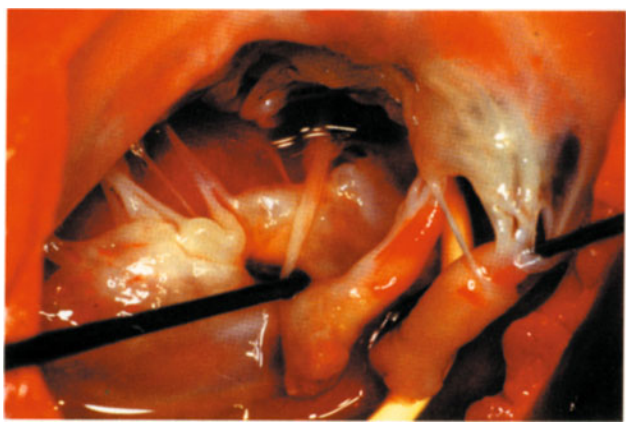


FIG. 30.3. Both ruptured papillary muscle heads in the right ventricle

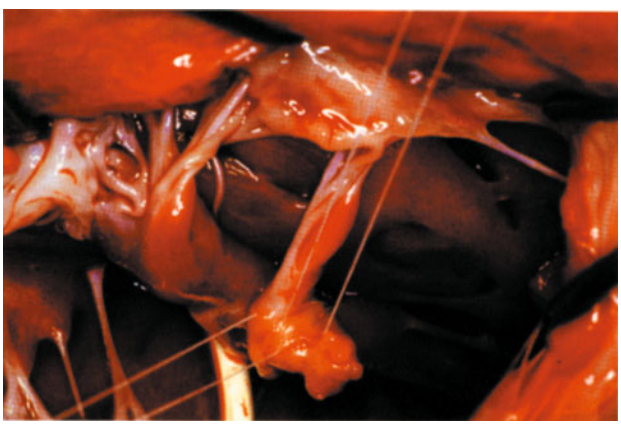


FIG. 30.4. The valve was repaired by reimplantation of both papillary muscle heads in the adjacent trabeculae using very fine 7-0 Gore Tex sutures

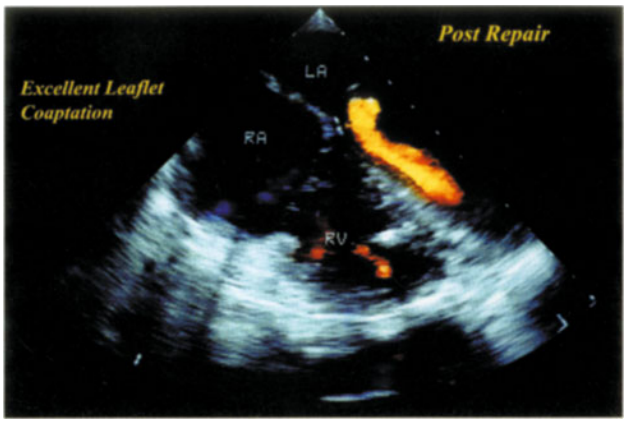


FIG. 30.5. TEE performed years later shows the perfect outcome of tricuspid reconstruction without regurgitation and with normal coaptation of the 3 leaflets

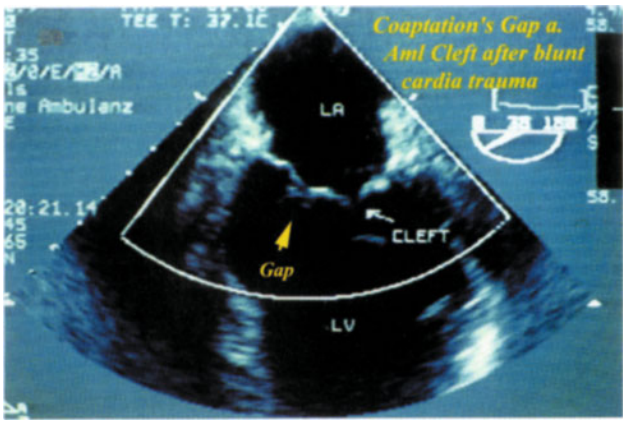


FIG. 31.1. A gap in the anterior leaflet on the 4-chamber view TEE in a 68-year-old woman after a road accident (so-called steering-wheel syndrome) that had occurred several years previously

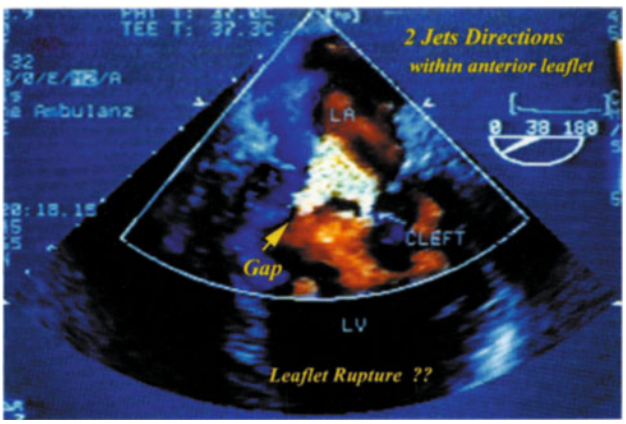


FIG. 31.2. Two different jet directions on color Doppler

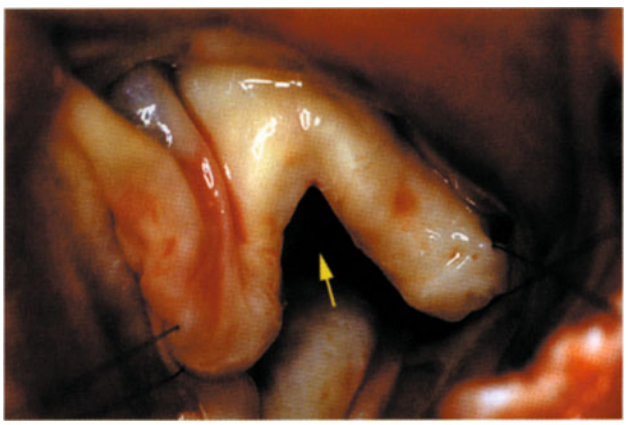


FIG. 31.3. The suspected rupture of the anterior leaflet was confirmed and it explained the jet phenomena: one jet is caused by the gap in the anterior leaflet, the other is due to the coaptation gap between the two leaflets after fibrotic shrinkage of the anterior leaflet as a result of traumatic leaflet rupture, which remained unnoticed for several years. This valve was repaired despite poor LV-function. An annuloplasty ring was placed additionally

remained unnoticed for several years. As a result of neglect (the patient had never had an echo examination after the accident to assess the reason for her dyspnea), the patient developed a markedly dilated heart with atrial fibrillation, flanked by pulmonary hypertension and dyspnea during mild stress.

The entire anterior leaflet was fibrotic and severely shrunken, which had caused a coaptation gap between the two leaflets. This pathology was readily visible in the TEE 4-chamber view on color Doppler preoperatively (Fig. 31.3).

The patient had a broad regurgitation jet in the coaptation gap, together with a narrow jet through the gap between the granulated edges of the anterior leaflet.

Severe shrinkage of the anterior leaflet and the cardiomegaly with severely impaired left ventricular function increased the surgical risk. After laborious reconstruction, the defect was closed

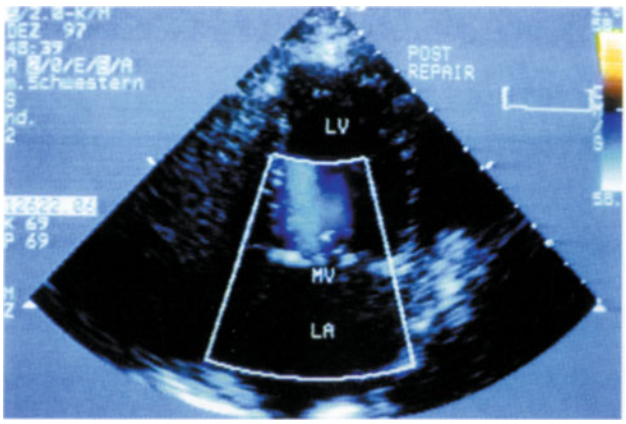


FIG. 31.4. Subsequent TTE showed a good outcome of reconstruction, with no regurgitation

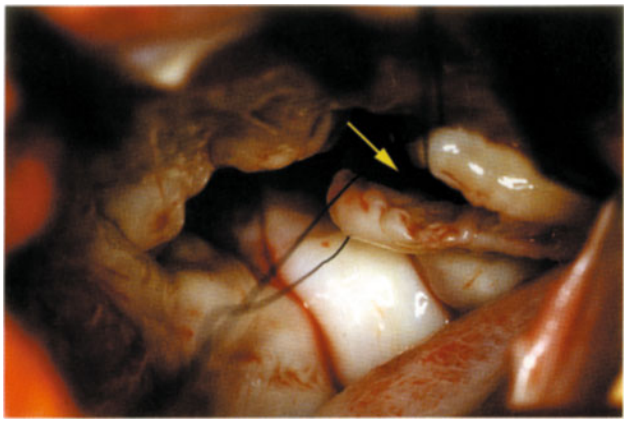


FIG. 31.5. Posterior leaflet rupture in a 23-year-old woman after a serious riding accident several years previously

with a patch of pericardium, combined with transposition of secondary chordae tendinae and placement of an annuloplasty ring. Although this led to a satisfactory functional outcome without mitral regurgitation (Fig. 31.4), the patient recovered very slowly and had to be kept in the intensive care ward for a long period of time. Subsequent echocardiograms still showed no marked reduction in the size of the heart or positive changes in terms of the functional dimensions of the left ventricle. Global cardiac function and the patient's quality of life, however, had improved.

This case shows that patients with previously restricted left ventricular function do not benefit much from mitral repair [30]–[32]. The indication for reconstruction should therefore be established early, before left ventricular function deteriorates and before atrial fibrillation develops [3], [9].

The postoperative recovery of a 23-year-old patient after a major *sporting accident* was much more favorable. The patient had had good left ventricular function before surgery. *The time span between the riding accident and the operation was tolerable*, viz. 2 years (in the intervening period the patient had given birth to a child). The ruptured site in the posterior leaflet, as shown in Fig. 31.5, still showed normal tissue structure and no more than a mildly granulated

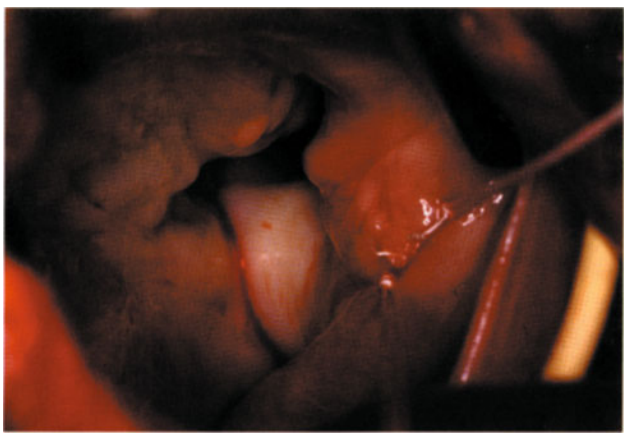


FIG. 31.6. The leaflet was reconstructed with inverted 7-0 Gore Tex sutures, before which the mildly granulated thickening in the lateral edge of the rupture was partly excised. In this young woman no annuloplasty ring was implanted. Instead, a few annulus compression sutures were used for additional security

thickening. As this patient did not have any annulus dilatation, implantation of an annuloplasty ring was not required. Thus, the patient was spared the discomfort of foreign material. Her leaflet was reconstructed only with a pair of 7-0 PTFE sutures converted inwards, prior to which the mildly thickened edge of the rupture was partially resected sideways (Fig. 31.6).

In valve disease caused by blunt cardiac trauma, **immediate surgery** should always be considered. Suddenly altered cardiac hemody-

namics may not be tolerated in all cases [30], [51], [54].

Following *pulmonary edema* after *an accident at work*, a 71-year-old farmer with a flail anterior leaflet (Fig. 32.1) was admitted to hospital. One week previously, he had received a blow from the hoof of a cow in his abdomen. Preoperatively, the modified long-axis TEE showed normal mobility of the posterior leaflet and no pathology in the annulus. This accurate diagnosis was an important element of the preparation for surgery and was of decisive importance for selecting the best repair technique. Experience has shown that reimplantation of the ruptured chordae tendinae is not ideal in such cases, especially when so little papillary muscle is available (Fig. 32.2). The plastic application of chordal transfer is always indicated in the presence of normal anatomy of the posterior leaflet, and was performed in this patient (Fig. 32.3). The 4-chamber TEE follow-up of this patient showed an excellent outcome after several years (Fig. 32.4).

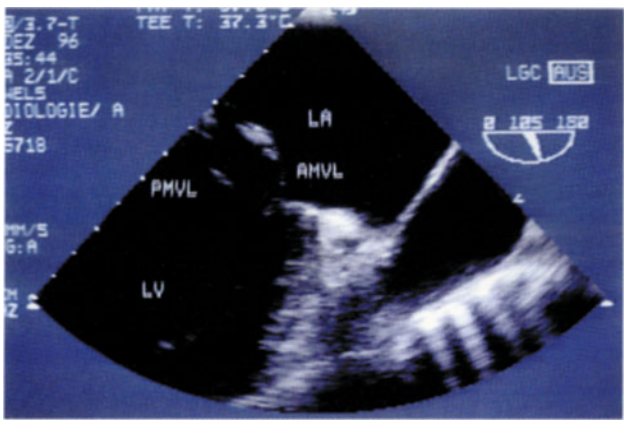


FIG. 32.1. Flail anterior mitral leaflet with normal mobility of the posterior leaflet on the modified long-axis TEE in a 71-year-old farmer after an accident (received a blow from a cow's hoof). An exact preoperative diagnosis based on echocardiographic examination is of decisive importance in order to apply the best possible surgical technique

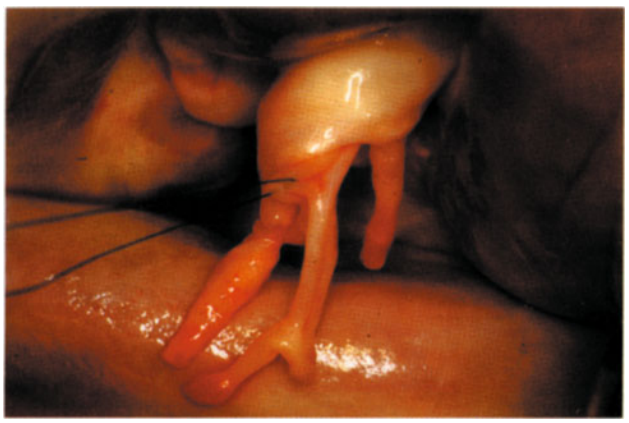


FIG. 32.2. At the operation this patient had rupture of the anterior leaflet chordae. The photograph shows the chordae tendineae with parts of muscle. The use of the flip-over technique for reconstruction is indicated in this patient because of the normal mobility of the posterior leaflet and the availability of sufficient chordae tendineae

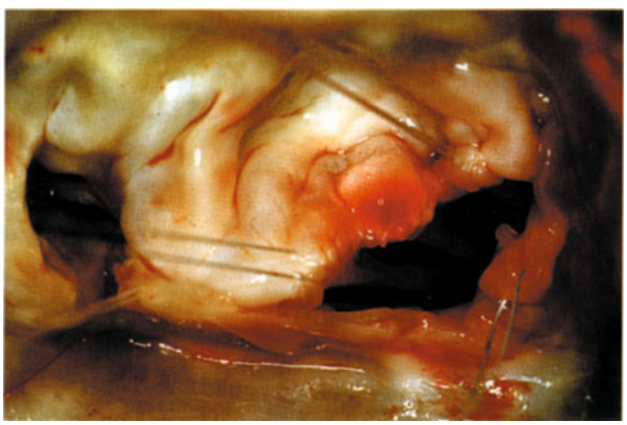


FIG. 32.3. Valvuloplasty after successful chordal transposition from the posterior leaflet to the anterior one, sutured with 7-0 Gore Tex

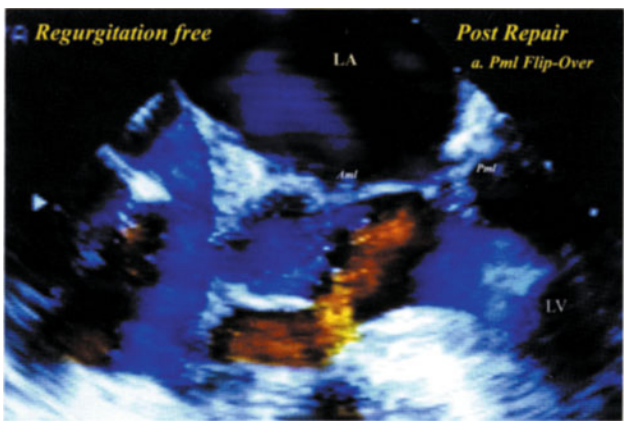


FIG. 32.4. Excellent postoperative result on the 4-chamber view TEE after several years, with no mitral insufficiency

Operations in the atrioventricular valves in the presence of blunt cardiac trauma

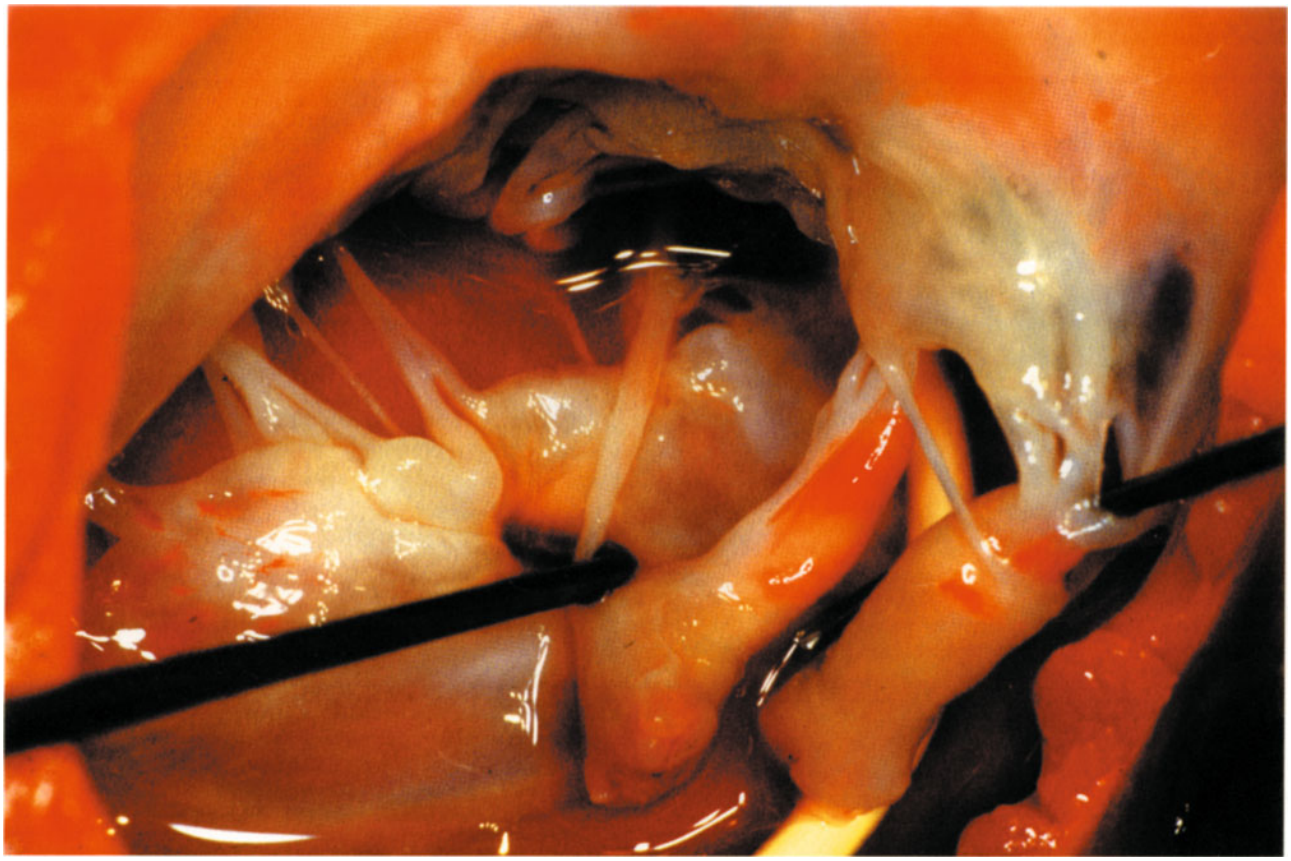


FIG. 30.3 (enlargement). Both ruptured papillary muscle heads in the right ventricle

Early reconstruction

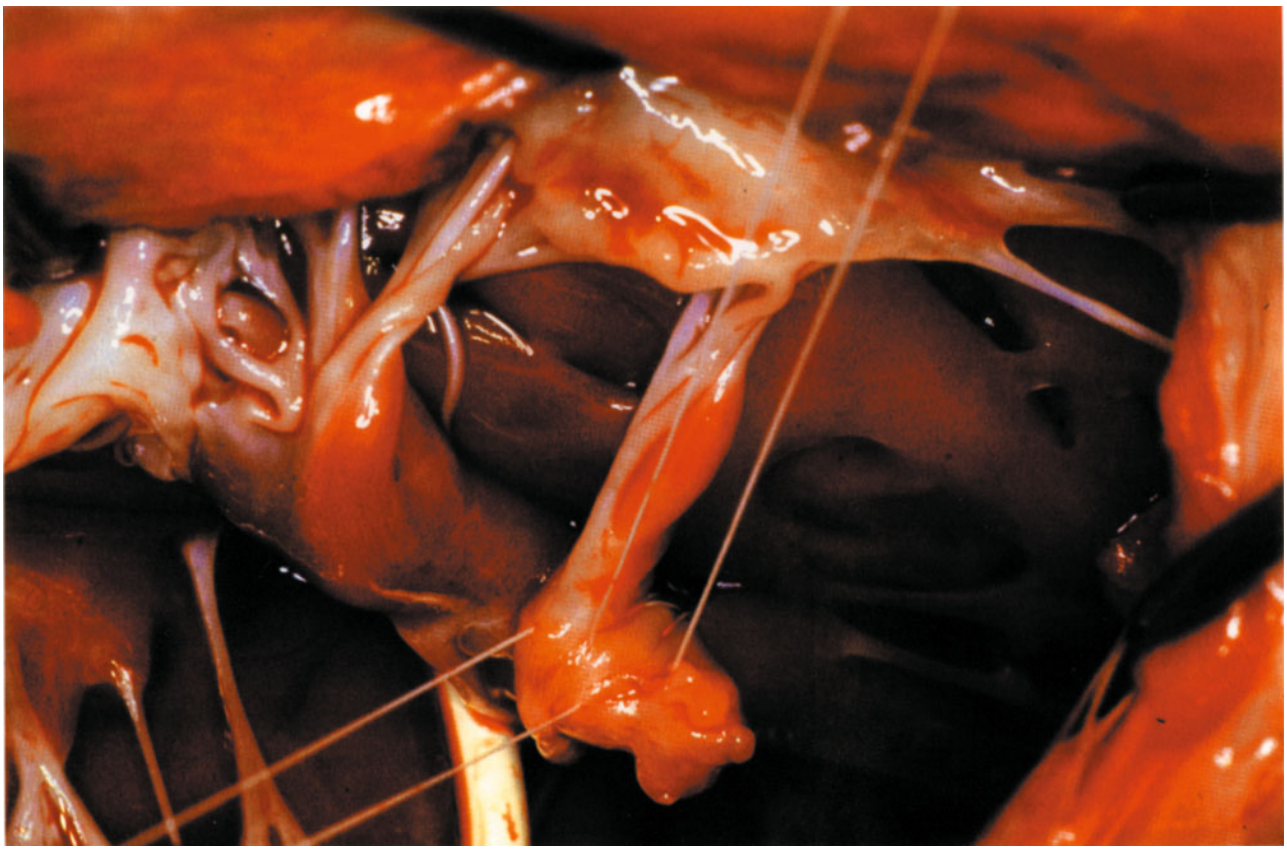


FIG. 30.4 (enlargement). The valve was repaired by reimplantation of both papillary muscle heads in the adjacent trabeculae using very fine 7-0 Gore Tex sutures

Operations in the atrioventricular valves in the presence of blunt cardiac trauma

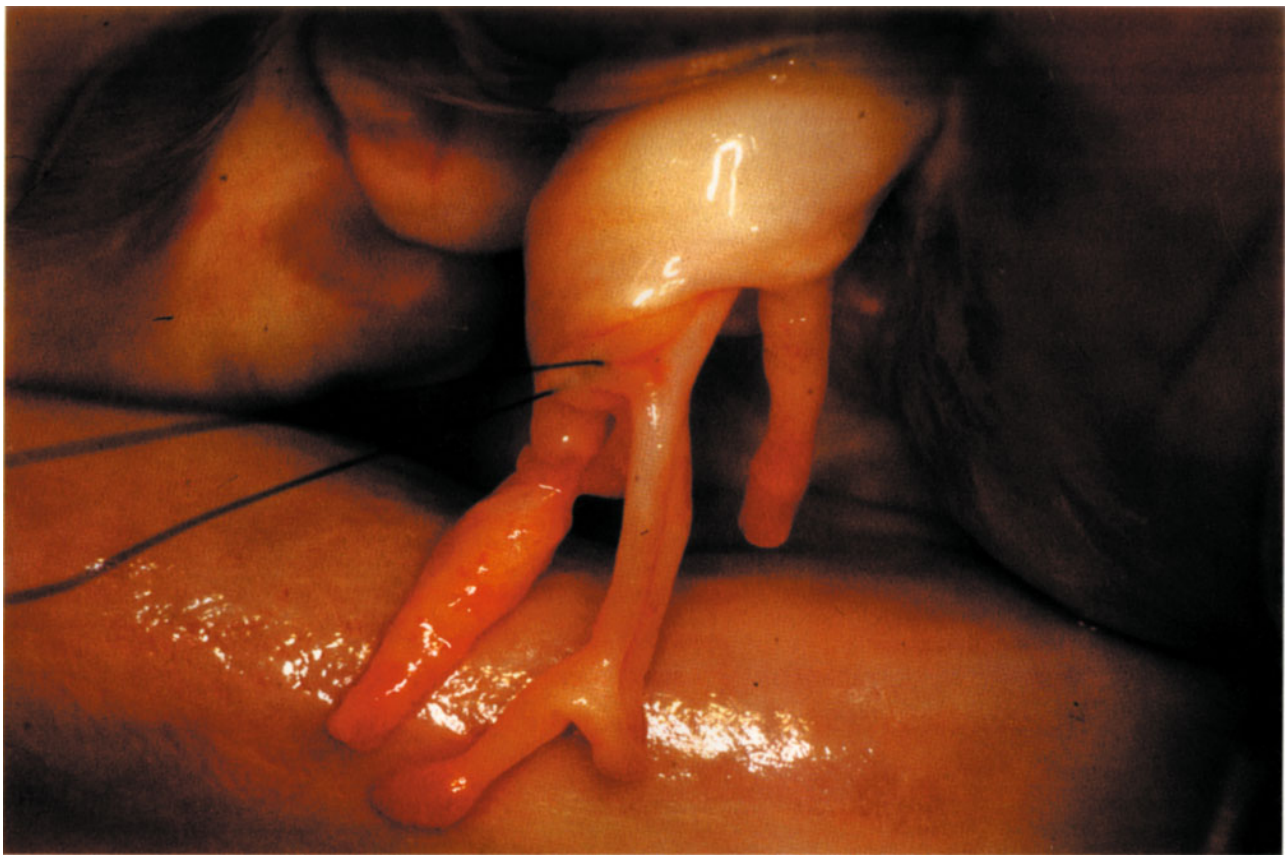


FIG. 32.2 (enlargement). At the operation this patient had rupture of the anterior leaflet chordae. The photograph shows the chordae tendineae with parts of muscle. The use of the flip-over technique for reconstruction is indicated in this patient because of the normal mobility of the posterior leaflet and the availability of sufficient chordae tendineae

13 Operations in the presence of multivalvular defects (Figs. 33 and 34)

13.1 Repair in the tricuspid valve in the presence of multivalvular defects

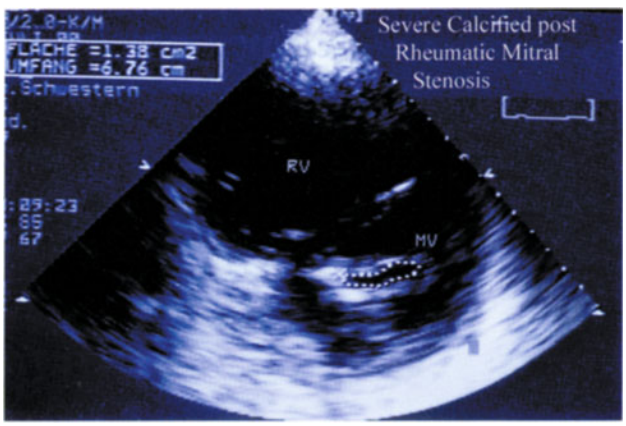
Currently, one occasionally encounters an organically altered rheumatic tricuspid defect with severe insufficiency. On the echocardiogram one finds severe fibrotic changes and coaptation gaps of the tricuspid valve.

Clear guidelines have been formulated regarding indication and treatment in severely calcified valvular mitral stenosis and aortic disease. However, organic tricuspid insufficiency is an entity that needs to be more clearly defined [1], [2], [9]. As long-term results of mechanical tricuspid valve replacement are markedly poorer than those after mitral and aortic valve replacement, valvuloplasty should most definitely be attempted in the presence of corresponding clinical symptoms and in the absence of severely calcified valves (Fig. 33.1–4). Surgical treatment with physiological approximation of the leaflets without permanent anticoagulation therapy has several advantages [5], [8], [9], [33], [55], [65].

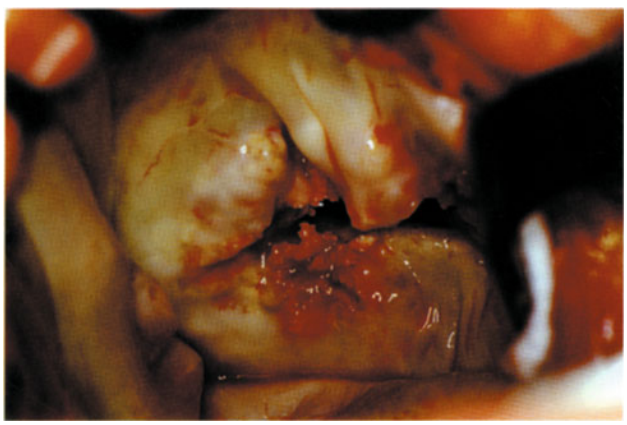
Repair procedures such as *De Vega annuloplasty* or implantation of a prosthetic ring at the tricuspid valve in cases of acquired valve defects are frequently performed today in the presence of fragile and unremarkable tricuspid leaflets with a normal suspension apparatus. However, as a result of a chronic mitral defect with pulmonary hypertension, the patient will have a markedly dilated tricuspid valve ring with secondary tricuspid insufficiency. As such tricuspid insufficiency may persist even after correction of mitral defects or a secondary tricuspid insufficiency may recur even several years after mitral valve replacement or in a patient with a functioning mitral valve after repair, it is advisable to perform this simple reconstruction procedure early [55].

13.2 Repair of the mitral valve in the presence of multivalvular defects

As soon as a symptomatic patient develops severe mitral valve regurgitation in conjunction with a moderate to **high-grade aortic valve stenosis**, a very serious situation may be considered to exist (Fig. 34.1–4) [33]. Urgent surgery is often indicated to prevent cardiac death [25], [26], [30], [31].

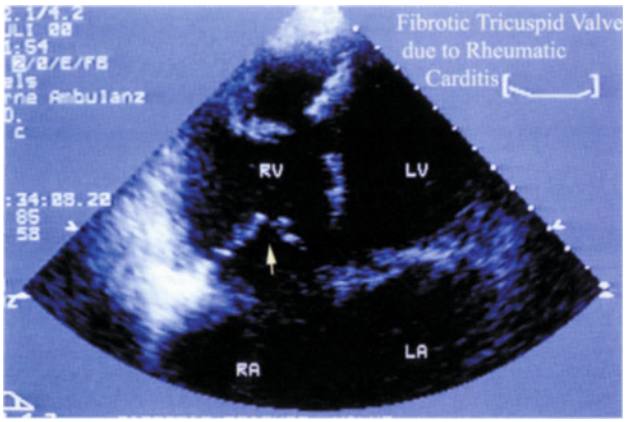


33.1

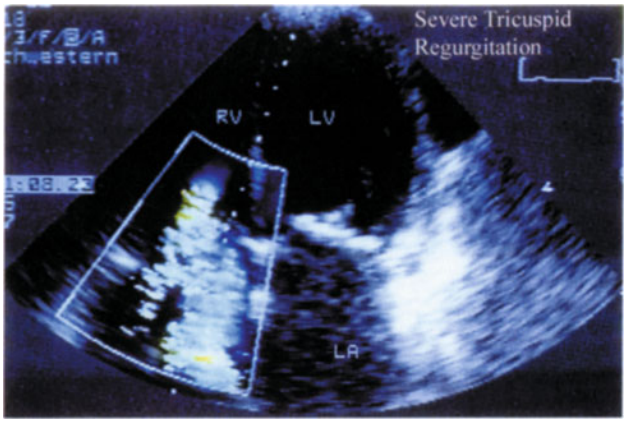


33.2

Fig. 33.1. TTE cross-section short-axis shows panvalvular calcifications with mitral stenosis. The surgical image shows stony calcification of the entire valve (**Fig. 33.2**)

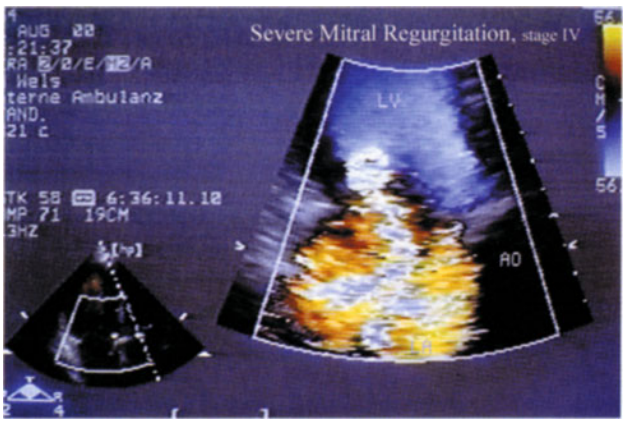


33.3

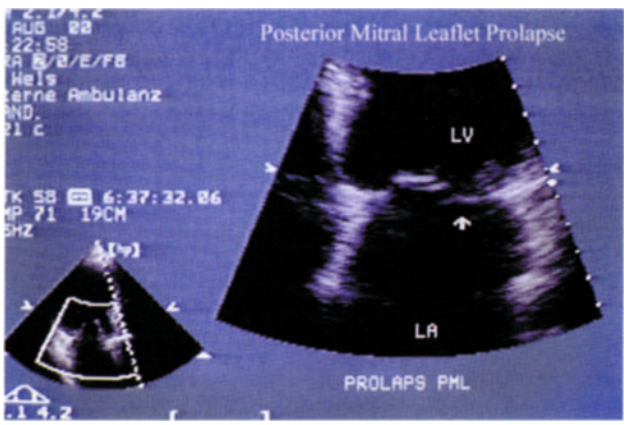


33.4

FIGS. 33.3 and 33.4. Extremely fibrotic tricuspid valve with a coaptation gap and most severe tricuspid regurgitation on the apical 4-chamber view. The patient was severely decompensated prior to surgery, with a pulmonary pressure of more than 60 mm Hg. In order to avoid late complications after double valve replacement, it is generally advantageous to preserve the tricuspid valve by mean reconstruction, last but not least, because the tricuspid position is not ideal for valve substitution



34.1



34.2

FIGS. 34.1 and 34.2. Severe mitral insufficiency secondary to posterior mitral valve prolapse (arrow) is seen in this color-Doppler image

Such combined defects are not uncommon even today in aged patients with degenerative valvular disease or after rheumatic endocarditis [1], [2]. When the decision to perform surgery is delayed, the outcome is rapid development of pulmonary edema or cardiac death after cardiac decompensation. Extreme pressure distension of tissue in the left atrium after cardiac decompensation has been observed to cause numerous tears in tissue, which makes surgery more complicated (Fig. 34.5, 6). *Delay in surgery* leads to a much poorer outcome of the operation and to increased intra- and postoperative mortality. In order to achieve better quality of life for these patients, early valve-conservation mitral surgery with aortic valve replacement using a bioprosthesis is a promizing approach. Especially patients with a preoperative sinus rhythm or those who have been converted to a sinus rhythm postoperatively profit from this modern treatment modality. Left ventricular function is improved after the procedure. This method of treatment also has the great advantage of being able to do without permanent anticoagulation [3], [4], [32], [56].

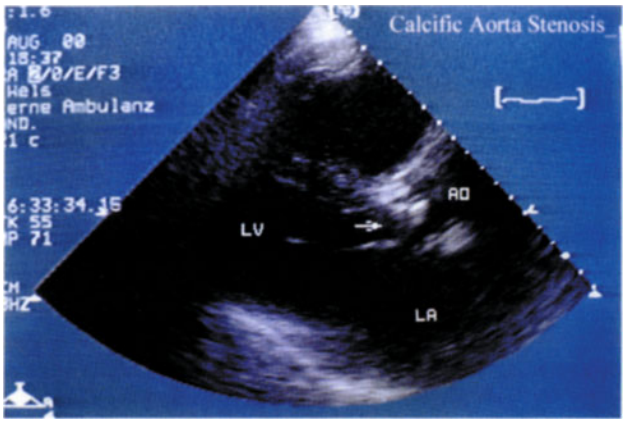


FIG. 34.3. Parasternal long-axis TTE view of the combined pathology, with severe aortic valve calcification

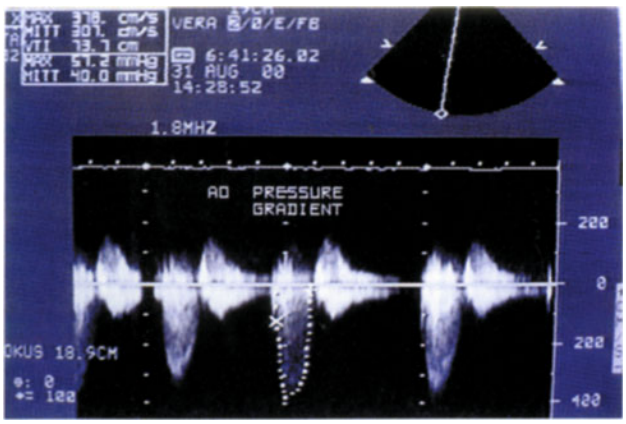


FIG. 34.4. Doppler recording across the aortic valve depicts high velocity and significant gradient in this patient with severe mitral regurgitation

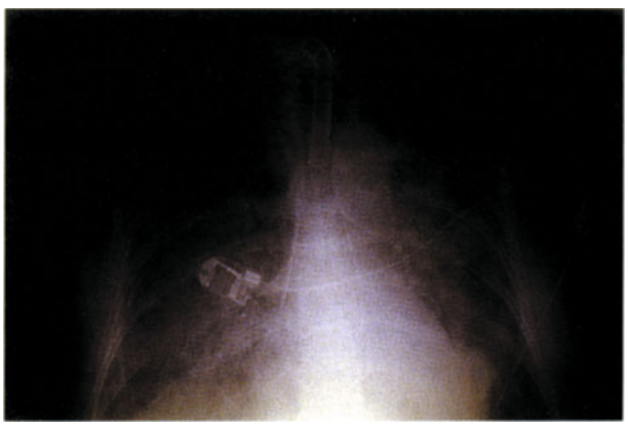


FIG. 34.5. A delay in surgery leads to pulmonary edema after cardiac decompensation and increases postoperative complications

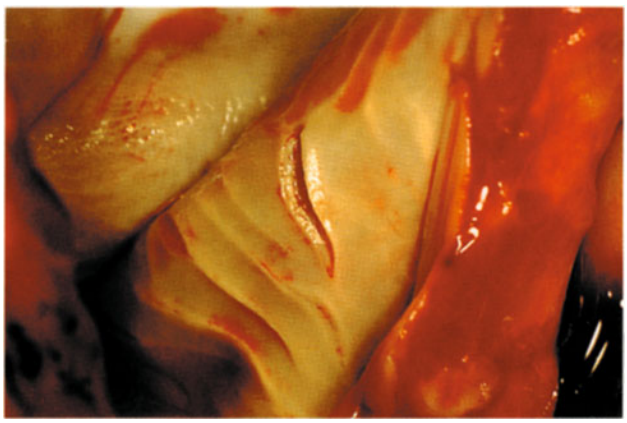


FIG. 34.6. Tears in the left atrial tissue due to extreme pressure distension of the left atrium tissue has been frequently observed after cardiac decompensation in such cases

14 Contraindications for reparative surgery

In the hospital at Wels, an average of 5% of patients with severe mitral insufficiency receive valve replacement due to contraindications for repair based on valve pathology, e.g. panvalvular fibrosis, calcifications in both commissures or calcification of more than 1/3 of the annulus, calcification of the anterior leaflet with spider-like stiffening of the chordae tendinae, multiple infectious destruction of the subvalvular apparatus and rupture of chordae in both papillary muscles. These patients are unsuitable for repair [5], [9], [11] (Figs. 35A.1 and 35A.2).

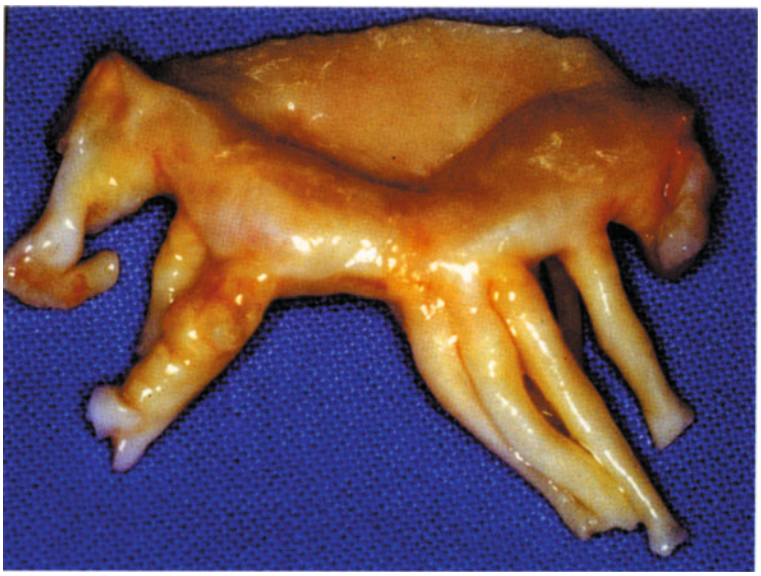


Fig. 35A.1. "***Spider-like***" *calcification of anterior leaflet*: bamboo-like stiffening of the chordae tendineae, severe fibrotic shrinkage of leaflet tissue with calcified anterior coaptation edge

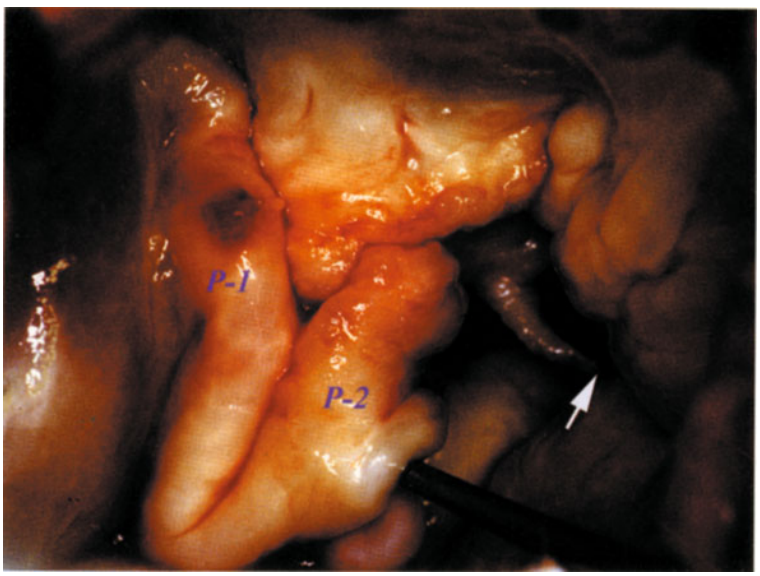


Fig. 35A.2. Post-endocarditis destruction of both leaflets, severe inflammatory tissue retraction and chordal rupture of anterior leaflet (white arrow), growth of vegetation in P-1 and prolapse along P-1 to P-2. This infectious pathology is not suitable for repair

15 Results

The various surgical methods of valvuloplasty and the diagnostic examinations described here have been used for 13 years in our institution in Wels. Of the 640 patients with mitral insufficiency who have been operated on so far, an average of 13% had a complex pathology. The innovative reconstruction techniques described above had been used in these patients. No intraoperative mortality was encountered in this group of patients with complex mitral valve pathology; nor have significant thromboembolisms or persistent neurological deficits been observed. The 5-year event-free survival is 90.5% [69]. The rate of reoperation (with repeat repair/valve replacement) was 4.9%. Two patients died within one year; one was due to renewed cardiac infarction and the other patient was due to acute cerebral vascular aneurysm. The postoperative echocardiographic studies in these patients showed improvement of LV function with a significant reduction of ventricular end-diastolic diameter and atrium dimensions [22], [49].

In the total population of 640 patients who underwent valve repair, the intraoperative mortality was 0.16% and the 30-day hospital mortality less than 1%, provided proper primary repair had been demonstrated postoperatively on echocardiography. The rate of reoperation with renewed valve repair was 0.57% and the rate of reoperation for valve replacement, 1.16%. One young patient with postoperative myocardial failure and severe LV hypertrophy was subjected to heart transplantation.

The previously mentioned mortality (*which was very low to start with*) was in no way associated with the complexity of the defects or with failure of the surgical technique. In all these cases, a routine operation (repair with quadrangular or triangular resection) had been performed in the presence of simple pathologies of the posterior leaflet. The advanced stage of myocardial damage with pre-existing strongly reduced LV-function (< 30% EF), complex dysrhythmia and other major co-morbid factors in the patients were decisive factors. Thus, it may be concluded that suitable patients should be offered the option of repair as early as possible.

16 Discussion

Surgical repair of the mitral valve has become the treatment of choice in select patients with clinically significant mitral regurgitation. Because appropriate patient selection for valve repair depends on the specific location and mechanism, a more detailed preoperative description of mitral pathology has become increasingly necessary.

Echocardiography has proven to be a valuable non-invasive and harmless technique that enables us to obtain valuable information about the pathology and function of the heart. Already in the emergency room the patient management can be evaluated without time loss. However, the quality of the transthoracic echocardiography TTE is sometimes limited due to various causes such as distance between the heart and the transducer as well as interference from the chest wall and the lungs.

Transesophageal echocardiography (TEE) has opened a new sonographic window to the heart. There is no interference from the chest wall and resolution is better as higher frequency transducers can be used. The nature of many structures can be studied better, improving the diagnostic accuracy. The indications for TEE are concluded from the advantages. If with TTE the images quality is poor, better pictures are usually obtained with TEE. The imaging capabilities of TEE make it ideally suited to assess mitral anatomy and pathology. In addition to the clinical features as well as the laboratory testings, echocardiography is the third important tool for infective endocarditis. This approach should allow regurgitant defects to be reproducibly located to that they can be followed throughout the course of the disease. Causes of the high resolution of TEE, changes of the valves can be detected before valve destruction has occurred. Thus, an early diagnosis and therapy seem to be possible and the prediction of prognosis is possible. Moreover, complications of infective endocarditis can rapidly be diagnosed. Because the left atrium is adjacent to the esophagus, therefore valuable information about the atrial contents is obtained with TEE. Arterial embolization and cardiac masses are good indications. The sensitivity and specificity as well as the predictive accuracy for TEE is so high, that it can not be challenged by computed tomography or angiography.

Routine intraoperative echocardiography approach without interference of surgical field has an important role in assessing the adequacy of repair, and thereby enhancing durability. In situations of initial unsuccessful valve repair, regurgitant jets can be localized and semiquantatively evaluated. During high-risk surgery, the left ventricular function can be monitored using the transgastric view. Using left ventricular monitoring; wall motion abnormalities can be detected earlier than ECG changes demonstrating myocardial ischemia and a reduced prognosis.

The real time three-dimensional echocardiography facilitates the study of structures of complex ventricular geometry. In clinical application, the possibility of computer slicing through the beating heart and the display of specific structures and the pathological conditions such as leaflet prolapse or calcification of mitral valve with three dimensional relation may greatly improve the diagnosis. This can provide a preview for the surgeon of what will be found during the surgery, which will be a great help in mitral valve reconstruction. Furthermore, accurate measurements of cardiac chamber volume and myocardial mass are feasible. In patients with rheumatic mitral regurgitation, three-dimensional echocardiography is able to show the malalignment of the papillary muscles and a narrowed interchordal angle that is opposite to the widening seen in mitral regurgitation from dilated cardiomyopathy. For the future, the three-dimensional echocardiography seems to be a promising tool for diagnostic decision. Certainly, this is a learning process, which requires skill and experience.

Mitral valve reconstruction comprises a number of surgical options to overcome the various mitral valve defects. Repair in cases of severe mitral insufficiency, complicated by complex mitral valve pathology such as extensive calcification of the annulus, infective destructive endocarditis, presence of abscesses, injury after blunt cardiac trauma, asymmetric geometry of mitral closure in ischemic disease, myxomatous degeneration, rheumatic endocarditis, combined and multivalvular defects, is strongly limited by the absence of diagnostic experience and a suitable surgical method on the one hand, and by the intact tissue remnants still available after aggressive tissue debridement on the other. In view of such situations, valve replacement is often given preference in cases of complex mitral valve pathologies. However, valve replacement also involves substantial risks, such as dehiscence of the valve prosthesis with paravalvular leakage, hemolysis, thromboembolism or hemorrhagic complications secondary to anti-coagulation. Also worthy of note are the well known disadvantages of valve replacement in comparison with valve repair. The former has unfavorable effects on left ventricular pump function and also affects postoperative morbidity and mortality, as a loss of the continuity of the valve annulus, the chordae tendinae and the papillary muscle during valve replacement eventually impairs the geometry of the ventricle. Preserving mitral valve geometry is the best preventive measure for postoperative preservation of left ventricular function [5], [8], [29], [51], [64], [67]. Reconstructive surgery in the mitral valve is associated with longer survival and lesser morbidity than valve replacement [3], [65], [66], [70]. The possibility of valve reconstruction should therefore be considered as a primary therapeutic strategy in all these patients.

Valve-preserving procedures

Myxomatous calcification of the annulus is a degenerative process that probably arises from mechanical stress on the mitral valve apparatus. Initially the calcification only involves the posterior mitral annulus, but then frequently spreads into the adjacent myocardium, the chordae tendinae and the papillary muscles (Figs. 3–8). This process must be distinguished from valvular calcifications that occur during rheumatic endocarditis and mainly involve the valve commissures. In the course of this disease, the annulus, starting from the thickened valve leaflets, is affected much later [17], [19], [26].

The removal of calcified masses as a part of reconstructive valvuloplasty in mitral insufficiency offers new treatment strategies. The complications associated with annulus calcification in mitral insufficiency, such as endocarditis, cerebrovascular attack, systemic emboli, conduction disorders and atrial fibrillation, and the surgical risk, can be reduced only by selecting the appropriate surgical method. Good long-term results have been reported when sufficient malleability and elasticity of the valve were preserved after mobilization of valve tissue and the subvalvular apparatus [3], [4], [8], [9], [19], [22].

The primary goal of surgery is to achieve extensive resection of calcified tissue and adequate mobility of the annulus as well as the valve leaflets. Severe calcification in the posterior annulus signifies a major risk for complications during valve-conserving surgery, especially in terms of rupture in the atrioventricular region. The use of pre-treated autologous pericardium (without antigenic side effects) in the form of endoventricular percardial patch annuloplasty is a remarkable technique for reducing the risk of ventricular rupture after extensive en bloc calcium debridement (Figs. 4, 6). In the mid term, it ensures good mobility of the valves [19]–[23].

The use of *untreated* autologous pericardium in mitral reconstruction has not been very successful so far, as it led to numerous post-implantation problems such as tissue retraction, fibrosis, the absence of smoothness, degeneration and endocarditis [57]. Pre-treatment of the pericardium is enormously important with regard to the durability of tissue and its resistance to calcification and degeneration in the long term [23], [36], [68].

The studies describing Carpentier's successful valvuloplasty procedures and the experimental results of Frater, Haluck and Love showed that treatment of autologous pericardium in a low-concentration glutaraldehyde solution should be not less than 15 minutes to preserve tissue stability and strength [59]–[61]. On the other hand, it should be no longer than 60 minutes to avoid excessive calcification. Briefly-treated pericardium appears to neither calcify nor shrink after implantation, as observed by Kumar et al [58]. The best long-term results after repair of congenital mitral insufficiency with this procedure were observed by Chauvaud et al. [36]. Scrofani et al reported excellent results without destruction of the transplant [62]. Preference was given to autologous pericardium as opposed to the standardized bovine pericardium. This factor probably contributed to the excellent results of repair achieved by Carpentier. Heterologous tissue was shown to be associated with the risk of virus transmission, including HIV-infection [63].

Glutaraldehyde-treated autologous pericardium is very suitable for combination with various techniques of valvuloplasty. The availability, malleability and processing potential of this material make it the medium of choice for correction of defects (Figs. 4, 6, 12–15, 20–23, 26, 29).

Most rheumatic patients with isolated mitral insufficiency who have lesions in the valve commissures and ulcerations of subendocardial calcifications in the left atrium usually have dilatation of the mitral annulus. Such dilatation is frequently encountered in cases of severe fibrosis and shrinkage in both mitral valve leaflets.

After removal of calcified masses in the affected valve commissure and the mitral annulus, successful valve reconstruction with full free mobility and closing valve leaflets can be achieved by means of **rotation paracommissural sliding plasty** (Fig. 8) or by a variation of valve repair derived from this procedure, using a pericardial patch, i.e., **paracommissural extension patch plasty** (Fig. 20.5, 6). Likewise, after extensive removal of calcifications with **sliding atrioplasty**, good adaptation of the posterior leaflet with attachment to the annulus and without any risk of incontinence can be achieved. Depending on the underlying pathology, severe shrinkage of the posterior leaflet is treated with **posterior leaflet extension patch plasty** (Fig. 22, Fig. 23.5). Valvuloplasty performed with the use of patches are stable and are not associated with early calcification or wear (Fig. 35.1, 2).

Our current experience in valve repair shows, together with the long-term results of other investigators, that successful correction can be achieved even in cases of severe calcification after myxomatous degeneration or rheumatic endocarditis. This is a major advantage in spe-

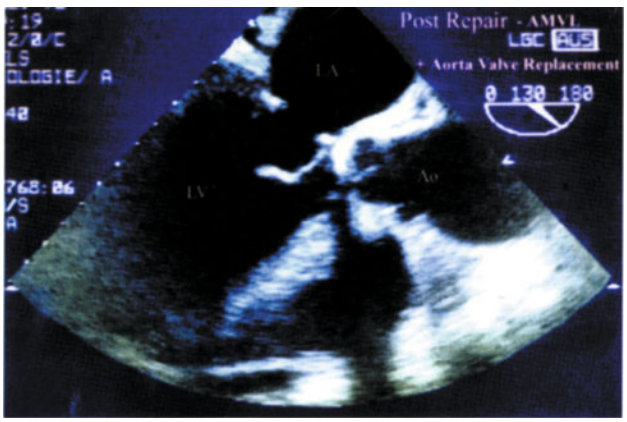


FIG. 35.1. TTE image in a 79-year-old woman with sinus rhythm who had undergone reconstruction of the anterior mitral valve leaflet and biological aortic valve replacement six years earlier. The anterior leaflet shows no tissue shrinkage after reconstruction. The patient enjoys excellent quality of life without anticoagulation; ventricular function has been restored

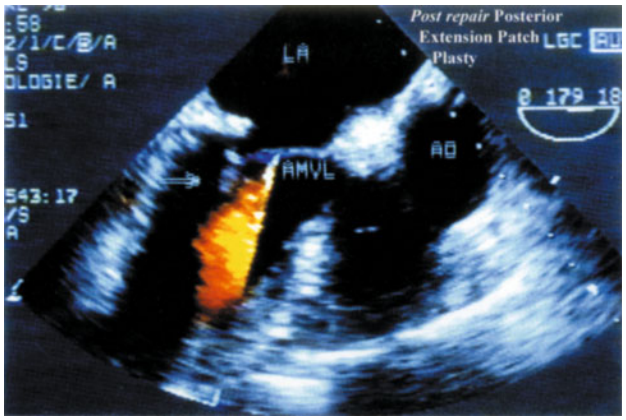


FIG. 35.2. Color Doppler TEE image in a 50-year-old woman eight years after reconstruction with leaflet extension plasty, The pericardial patch in the posterior leaflet shows no significant leaflet calcification and there is still excellent re-coaptation of both leaflets without regurgitation

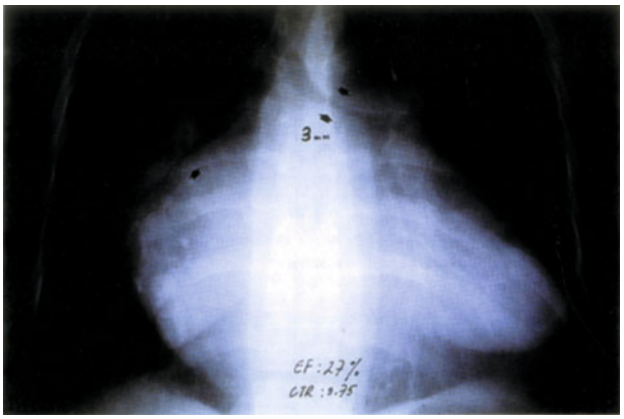


FIG. 35.3. Preoperative Chest X-ray of a 25-year-old woman with severe rheumatic mitral valve insufficiency due to post-rheumatic carditis. The arrows show the compression of both main bronchi due to dilatation of the atria. Left ventricular ejection fraction was 27% preoperatively

cific groups of patients in whom anticoagulation should be avoided and LV-function preserved as well as possible.

I present the example of a young woman with rheumatic endocarditis.

A 25-year-old Nigerian woman was admitted with severe mitral valve insufficiency in stage IV (type III). With an ejection fraction of 27%, pulmonary hypertension, cardiomegaly and a heart-thorax ratio (HTR) of 0.75, the patient had severe cardiac decompensation. She also had high-grade ascites, peripheral edema and severe respiratory obstruction due to bilateral impression of the main bronchus as a result of massive dilatation of both atria. The diameter of the main bronchus measured, was only 3 mm (Fig. 35.3). Six months after successful valve reconstruction, her heart had recovered impressively from the insufficiency (HTR was markedly reduced to 0.57 while EF had increased to 57%). By reducing the dimensions of the atria, the impression on the main bronchus was relieved. The diameter was nearly normal (9 mm) when the patient was discharged. She returned to her country free of clinical symptoms and without permanent anticoagulation (Fig. 35.4).

Obviously, the possibilities of mitral valve reconstruction are not unlimited. Patients with

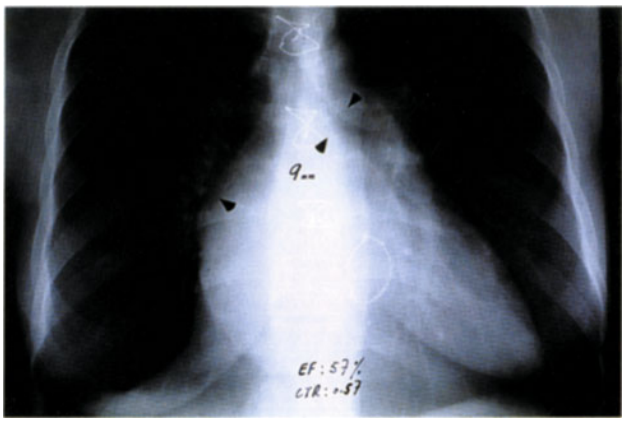


FIG. 35.4. Chest X-ray six months after successful valve reconstruction displays striking reduction in cardiac size. The patient became asymptomatic. Her left ventricular ejection fraction had increased to 57%, remained in sinus rhythm without need for anticoagulation, and she was symptom-free

rheumatic or infective endocarditis have a higher rate of reoperation. Recurrence of the disease process is related to its complexity and to the involvement of various portions of the mitral valve apparatus. However, the type of reconstructive procedure and the experience of the surgeon may influence the subsequent course. The concept of comprehensive surgical correction for achievement of good valve function with

a good long-term outcome should be directed towards repairing all diseased components of the mitral valve apparatus. Different innovative techniques of reconstruction are used to achieve satisfactory results in mitral valve repair.

In general, mitral valve repair has been considered a contraindication during the **acute phase of endocarditis**. As a result of valve infection, various pathological conditions with destruction may occur (e.g., valve vegetations, leaflet perforations, chordal ruptures, annular abscesses and fistulae). Transesophageal echocardiography is invaluable clinically since it accurately images the extent of valvular destruction or the associated abscess cavities. It provides the information about the site and extension of abscess, which is needed by surgeons to time and define surgical intervention. Early diagnosis with application of TEE is advantageous, and abscesses should be expected in 5%–25% of patients with echocardiographically discernible vegetations. There is no abscess typical organism; therefore transesophageal echocardiography examination should be applied at the initial suspicion of infective endocarditis, to identify high-risk patients. Multivalvular involvement, rapid growth of vegetations and the presence of spontaneous echo contrast are echocardiographic indicator of high-risk endocarditis. Factors strongly associated with poor outcome in patients suffering from infective endocarditis are congestive heart failure, severe valvular regurgitation and uncontrolled infection, such as paravaluvlar abscess formation and intromyocardial spread. Transesophageal echocardiography markedly improves the detection rate of abscesses associated with endocarditis. Since patients with infected native valves and associated abscesses have a worse prognosis having a high mortality rate between 40%–90%, they should be considered as at high risk [27]–[29]. Surgery can reduce this mortality. However, an extensive lesion with insufficient material for reconstruction usually prompts the surgeon to favor mitral valve replacement. Although mitral valve replacement has drastically improved the results after acute endocarditis, the implantation of prosthetic material in infected tissue remains a questionable procedure. The rate of infection after prosthetic valve replacement is reported to be as high as 8 to 20%, and reinfection usually involves the same organism cultured at the initial valve replacement [71]. Creative valvuloplasty with a pericardial patch produces good results, even if aggressive excision of infected tissue needs to be performed. Achieving the goals of surgery require the removal of vegetations, which are potential risk factors for systemic emboli. Abscess cavities must be rinsed and closed, as they may develop into foci that are resistant to antibiotics. Fistulae between the two ventricle or other structures must also be closed. Restored valve function leads to optimal hemodynamic results.

The previously described **sliding atrioplasty** (Fig. 7), **leaflet sliding plasty** (Fig. 10), **patch as substitute for leaflet** (Figs. 13–15), **sliding plasty with chordal shortening** (Fig. 24), **chordal transposition plasty** (Fig. 25) and **chordal replacement with a narrow strip of patch** (Fig. 26) are relatively uncomplicated and suitable for practical application. The risk of the SAM (systolic anterior motion) phenomenon after mitral repair, especially in the presence of *Barlow's disease*, does not exist.

Rotation paracommissural sliding plasty is characterized by the creation of a neo-commissure that arises as a result of extensive sliding and rotation maneuvers of the posterior mitral valve leaflet. Careful technical execution produces a tightly fitting valve with a sufficiently large opening surface of the valve while optimally utilizing the remnants of valve leaflet tissue and the corresponding chordae tendinae. The goal should be to achieve a 1:3 size ratio between the posterior and the anterior mitral leaflet.

In cases of active infective endocarditis with extensive vegetations in the anterior and posterior leaflet and involvement of the commissures, one needs to perform extensive resection in both leaflets (Fig. 20.1, 2, and 5). In such cases, *rota-*

tion paracommissural sliding plasty alone is not enough. To preserve the valve, additional autologous pericardial tissue is needed. The autologous pericardial patch serves as substitute tissue and bridges the gap between the detached paracommissural annulus and the re-coaptation surface of the two leaflets (Fig. 20.6). Even *slight tension* between the tissue of the two leaflets and the annulus after repair would lead to renewed valve insufficiency secondary to dehiscence or chordal rupture [11]. Therefore, the use of a pericardial patch together with rotation paracommissural sliding plasty is the ideal solution for *tension-free repair* in the treatment of a complex commissural pathology [23], [24], [49].

The early echocardiographic identification of infective pathology accompanies with application of a variety of repair techniques permit successful results, even in cases of severe mitral valve destruction by infection. We have achieved in this severely ill patient group – namely, no early mortality, no late cardiac related mortality and currently no reoperations associated with recurrent endocarditis.

If mitral regurgitation is the result of **ischemic necrosis of the papillary muscle** and the adjacent ventricular areas (Fig. 29A.1–3), a surgical intervention in the presence of poor LV function signifies high morbidity and mortality. Although mitral repair should be given preference in this setting, most surgeons replace the valve. Mitral valve replacement in chronic ischemic mitral regurgitation is associated with a 30-day mortality as high as 15% [5], [9], [72]. The most important reason for the unpredictability of the outcome of repair is the asymmetric geometry of the mitral valve leaflet, followed by papillary muscle dysfunction and infarction of the adjacent myocardium, or annular dilatation after primary infarction with subsequent LV-dilatation. The infarcted papillary muscle no longer contracts and is progressively shortened, as the necrotic muscle is replaced by fibrous tissue. In end-systole, the apical portions of papillary muscles are not uniform and show an asymmetric shift of dysfunction. These distortions limit leaflet mobility, which causes reduced leaflet coaptation. The discoordination of papillary muscles in conjunction with annular dilatation leads to distortion of valve closure. In echocardiographic studies, the infarcted posterior left ventricular wall bulges outward in systole and drags the posteromedial papillary muscle away with. The tethering forces on the chordae tendineae emerging from the affected posteromedial papillary muscle are increased, resulting in restricted posterior mitral leaflet motion corresponding not only to medial half of the middle segment but also involving the entirety of the posteromedial commissure. This results in a incomplete coaptation of the anterior and the posterior leaflet and thereby severe mitral regurgitation.

Ischemic mitral regurgitation is an extremely common complication of myocardial infarction and significantly reduces short- and long-term survival. Valve repair with direct suture between the anterior mitral leaflet and posterior mitral leaflet at the posteromedial commissure (A3 to P3), commissuroplasty, has been tried in chronic ischemic mitral regurgitation with varying success. Although earlier studies dealt with the subject of preserving valve function with an annuloplasty ring, this procedure is unfortunately not very efficient (in terms of eliminating severe mitral insufficiency in all patients) [39], [41], [46], [47]. This surgical technique used currently in chronic ischemic mitral regurgitation is "downsizing" of the mitral valve with mitral annuloplasty rings that are one to two sizes smaller than would have normally been applied. Isolated annuloplasty fixes ischemic mitral regurgitation, because it compensates for the posterior left ventricular wall dilation. The papillary muscle is moved down and the ring brings it together and achieves competence. However, this procedure transforms the mitral valve into a "monocuspid valve", with the anterior mitral leaflet as the sole functioning leaflet. This annulus remodeling technique ought to produce further restriction of the entire posterior leaflet, and, in time, lead to progressive increase in the

tethering forces on the chordae and papillary muscles. Moreover, this repair method may complicate the valve function in relation of affecting adversely both early and med-term results. It has been reported that a significant number of patients develop recurrent mitral regurgitation at intermediate follow up, because LV in some patients continue to dilate despite the coronary revascularization and the ring annulopasty procedure. Patients presented with marked annulus dilation and impaired left ventricular function – resulting in leaflet restrictive motion had significant mortality with isolated ring annuloplasty treatment. Mitral ring annuloplasty alone in patients with chronic ischemic MR and more then 2+ MR has been shown to be attended with a significant residual MR in 10% to 28% of patients [73].

Our experience in terms of the results of previous investigations concerning repair confirm those of Komeda and colleagues [43], who showed that the asymmetric displacement of the papillary muscles after infarction could not be normalized with an annuloplasty ring alone. Repair of valves with this asymmetric geometry requires also readjustment of the leaflet excursion or the papillary muscle–annular relationship. With the papillary-chordae length unaltered, the coaptation gap or incomplete mitral leaflet coaptation due to asymmetric geometry can be corrected by increasing the posterior leaflet surface area using extension plasty with autologous pericardium (Fig. 29A.5–8, Fig. 29B). The leaflet edges are brought closer together, thereby allowing coaptation to occur more readily. This new strategy, in addition to utilizing an annuloplasty ring to reduce the annulus dilatation, successfully eliminated ischemic mitral regurgitation in our patients with restrictive leaflet motion. When the results of leaflet extension plasty are good, this is likely to have readjusted the muscle-annular relationship of a geometrically deformed mitral valve after acute or chronic myocardial ischemia [45], [49]. The patch enlargement repair technique in chronic ischemic mitral regurgitation with restricted posterior mitral leaflet motion offers an anatomic and physiologic correction of the underlying defect and should be considered as an alternative to other currently practicable surgical methods. Leaflet extension plasty can offer a better long-term outcome [74].

Moderate mitral regurgitation remains a problem that is approached differently depending on individual cardiologist or surgeon opinion. Because of the variability of the symptoms and the findings at echocardiography and at heart catheterization, ischemic mitral regurgitation may be easily overlooked, we must consciously search for it. A very careful assessment of the ischemic mitral valve pathophysiology by preoperative and intraoperative transesophageal echocardiography is essential for a correct decision-making process. A strategy for ischemic MR treatment has been adopted [46]. Currently, preoperative stress echocardiography and intraoperative dynamic testing have essentially helped us to identify the pathophysiology or mechanism, in which a definitive valve procedure added to a complete myocardial revascularization will be decided. This performance can minimize the mortality of patients.

The majority of patients who develop *cardiogenic shock* following myocardial infarction characterized by acute or progressive hemodynamic deterioration may result from rupture of the ventricular septum, left ventricular free wall, and papillary muscles. Myocardial rupture often occurs in patients with small areas of infarction and well-preserved systolic function, as shear stress increased in the area of necrosis or its boundaries leading to the mechanical disruption [75]–[77].

The prognosis of acute papillary muscle rupture associated with myocardial infarction before surgical intervention was poor, with 33% of patients dying immediately, 50% dying within 24 hours. In patients presenting in shock, mortality with medical treatment alone is inevitable [78].

The two separate papillary muscles, the anterolateral and posteromedial papillary mus-

cles share the innervation of the contiguous left ventricular wall and have their appropriate position in the contraction sequence. The posteromedial papillary muscle typically consists of one or two large common trunks and multiple heads, all of which give off chordae to both the anterior and posterior mitral valve leaflets. Following an acute inferior myocardial infarction, it is the posteromedial papillary muscle that is most vulnerable to infarction, necrosis, and rupture. This vulnerability has been related to the fact that its blood supply usually is dependent entirely on the coronary artery that supplies the crux of the heart, whereas the anterolateral papillary muscle is often in a watershed area between two major coronary arteries and is supplied by both arteries. Rupture of the entire or large common trunk will result in a severe mitral regurgitation or immediately to death (Fig. 27.1–2, Fig. 28.1–2), which is typically characterized by acute rapid haemodynamic deterioration presented with severe dyspnoea and raised venous pressure usually several day after an infarction. In these desperately ill patients, pulmonary edema is almost always present or severity of mitral regurgitation, such as left or right ventricular dysfunction, may result in cardiogenic shock. Complete rupture of one of the multiple heads of the papillary muscle causes less severe deterioration than rupture of the entire trunk; however, the potential leading rapid decompensation is predicable, even after initial stabilization with medical treatment (Fig. 27.3).

The sudden development of congestive heart failure or shock in a patient following an acute inferior myocardial infarction with a previously stable condition should suggest the diagnosis of a mechanical complication including papillary muscle rupture, and trigger rapid evaluation and expeditious surgical management of the patient.

Acute mitral regurgitation and papillary muscle rupture should be suspected by the presence of cardiogenic shock or pulmonary congestion with or without systemic hypoperfusion in patients.

The typical patient with ruptured papillary muscle is mostly in the first week after inferior myocardial infarction. The diagnosis of papillary muscle rupture in our patients is generally made rapidly and non-invasively by echocardiography. A transthoracic echocardiogram can visualize directly a flail mitral leaflet or swinging papillary muscle. In those patients in whom the mitral apparatus cannot be directly visualized, the diagnosis is suspected by the presentation of hemodynamic compromise concomitant with preserved systolic function, in which case transesophageal echocardiography can be performed for further delineation (Fig. 27.2, Fig. 28.1).

Proper recognition and management of the emergencies in mitral valvular disease can be particularly gratifying. Of all the ruptures that be found after inferior wall infarction, it is the patients with papillary muscle rupture who will benefit most from an early surgical repair diagnosed within hours of presentation. Because the correction of the mechanical complication may leave the patient with relatively intact ventricular function, which in turn may be a powerful predictor of a favorable long-term outcome. These patients will not only survive surgery but also enjoy a better life in the years to come.

Reconstructive mitral valve surgery without the use of **a ring** is associated with a high rate of early failure [34], [36], [48], [79]. Thus, annuloplasty is the prerequisite for successful and sustained valve repair. The ring serves to consolidate the valve repair and to re-form the annulus as well as to ensure the integrity of the mitral annulus. Modern concepts of valvuloplasty require a combination of neo-formation of the mitral annulus to avoid tension in the reconstructed valve leaflet on the one hand, and flexibility of the mitral annulus to preserve physiological valve function on the other [34], [48], [79]. Full flexibility of the posterior segment of the annulus permits physiological three-dimensional mobility and also has a very positive effect on the fragile atrioventricular tissue after annulus decalcification. Stabilization of mitral valve repair is achieved, which reduces

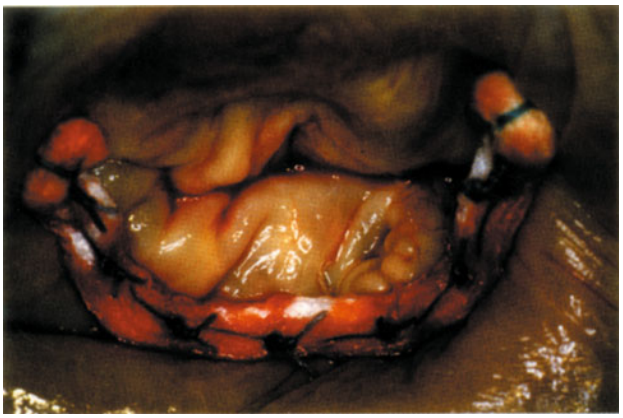


FIG. 36. Flexible posterior annuloplasty band (Cosgrove-Edwards(R) Annuloplasty System)

tension at the sutures and also minimizes the risk of dehiscence. Routinely, ring annuloplasty accompanies all repairs with exception for high-risk cases of infective endocarditis. In no patient so far did we encounter the phenomenon of systolic anterior motion (SAM) with partial obstruction of the left ventricular outflow tract following implantation of a rigid ring or flexible posterior annuloplasty band. According to our present observation in patients with degenerative disease, a flexible posterior annuloplasty band (Fig. 36) provides results regarding to repair durability or patients survival equivalent to those obtained with a rigid or flexible circumferential annuloplasty ring. We believe, flexible annuloplasty has theoretical advantages; but long-term studies should prove its clinical benefits.

In our surgical strategy, for such cases of infective endocarditis, we perform modified techniques for mitral valve repair without ring annuloplasty in order to avoid the use of too much foreign material [83]. This may have advantage to prevent recurrent endocarditis, which contributes to our good results.

Currently, no ideal prosthetic substitute for the mitral valve is available. Therefore, strategies aimed at preserving the patient's own valve and avoiding permanent anticoagulation are very attractive. Carpentier, Duran and Bernal describe concepts that are based on reconstruction of all diseased portions of the mitral valve with the goal of achieving good valve function in the long term [36], [65], [66], [80]–[82]. Improved surgical and technical prerequisites and advances in drug therapy have led to excellent results in the last few years and to a substantial reduction of complications. The cases reported here represent my experience with innovative valvuloplasty in patients with complex pathologies of the mitral valve and also include the "learning curve".[1]

Delaying the application of mitral valve surgery in mitral insufficiency often leads to irreversible damage to left ventricular pump function [31], [32], [84]. Very early valve repair may be regarded as an aggressive therapeutic procedure, but does lead to better early and long-term results, especially as the presence of a complex valve pathology in mitral insufficiency is a marker of poor prognosis in the spontaneous course of the disease [3], [4]. Provided mitral valve repair is performed by experienced surgeons who are familiar with the new techniques, an improved outcome may be anticipated. Early application of surgical measures will create new therapeutic and management strategies for these patients even before complications like atrial fibrillation, left ventricular dysfunction or cardiac insufficiency can occur.

Good results can only be achieved when it is ensured that several factors are present simultaneously:

(1) Preoperative detection and accurate assessment of the pathology with the help of echocardiographic imaging.
(2) Reliability of the operative techniques.
(3) Modern postoperative care.

[1] (Part of the results of reconstruction in patients with complex mitral valve pathologies were published in The Annals of Thoracic Surgery in July 2000 and January 2001. The publications have been selected as Papers for Discussion in the Internet) [22], [49].

17 Conclusion

With innovative repair techniques, mitral valvuloplasty can be safely and successfully performed even in the presence of complex mitral apparatus pathology [22], [49], [56], [77], [79], [80]–[82]. Our experience demonstrates good and stable results after valvuloplasty in cases that would otherwise have been subjected to valve replacement. The ultimate goal apparently is restoration of a "neo-valve" with normal valve function while avoiding the use of foreign materials. Modern techniques of surgery described here, in conjunction with precise echocardiographic evaluation, will aid in meeting these requirements. The methods of surgery are well reproducible and can be performed within a short period of time. They permit either long-term or definitely successful valve repair. Our success in conjunction with low intra- and perioperative complication rates and the good long-term results should serve as an encouragement to apply mitral valve surgery very early – even before left ventricular dysfunction occurs.

In cases of complex valve pathology with severe hemodynamically significant mitral insufficiency, the surgeon and cardiologist caring for the patient should inform the latter of the possibility of valve repair. With the knowledge of precise interpretation of echocardiography and application of innovative valvuloplasty techniques, mitral valve repair may already be regarded as the surgical method of choice today for mitral valve disorders [18].

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Fig. 23.1. The mitral valve orifice area (MVA) in a 35-year-old woman with rheumatic mitral stenosis with failed balloon dilatation measured 0.9 cm^2; **Fig. 23.2.** Doppler recording discloses a transvalvular pressure gradient of 10 mm Hg and also mitral regurgitation

Fig. 23.3. The surgical analysis shows valvular stenosis and the mechanism for insufficiency. Primary congenital commissural fusion with subsequent rheumatic disease was causative for severe valvular fibrosis and shrinkage of leaflet tissue in this patient. All main chordae and the corresponding papillary muscle heads were extremely stiff and retracted, but not calcified

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Fig. 25A.4. After successful transfer of the posterior chordae tendineae to the anterior leaflet

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Fig. 25B.2. One group of the chordae tendineae has been attached to the coaptation edge of anterior leaflet

Secondary chordae tendineae are used in modern mitral valve reconstruction of healthy tissue with normal subvalvular length. Therefore, after successful chordal attachment to the coaptation edge where support is needed, this will help eliminate mitral regurgitation due to chordal elongation or rupture

Fig. 26.1. The upper arrow on the long-axis TEE shows a flail anterior leaflet, the lower arrow shows the posterior annulus calcification in a 74-year-old woman with reduced left ventricular function without atrial fibrillation

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Fig. 29A.2. Color Doppler shows severe excentric mitral regurgitation

Fig. 29A.3. Infarction of the papillary muscle and the adjacent myocardium leads to shrinkage and shortening of the suspension apparatus of the mitral valve. This results in limited mobility of the mitral leaflet and incomplete mitral leaflet coaptation. Myocardial infarction may lead to annulus dilatation and, in severe cases, to global dilatation of the left ventricle. Annulus dilatation, coaptation gaps of the mitral leaflet and discoordination of the papillary muscles eventually lead to severe mitral insufficiency

Figs. 29A.4–8. Valvuloplasty, with restoration of valve mobility and the relationship between papillary muscle and annulus after free mobilization of the fibrotic papillary muscle, is achieved after enlargement of the surface of the posterior mitral leaflet by means of leaflet extension plasty with autologous pericardium. The leaflet edges are approximated to each other so that coaptation is achieved. Anchoring of an annuloplasty ring is obligated to ensure correction of annulus dilatation and to fecilitate improved coaptation of both leaflets so that mitral regurgitation

Fig. 29B.1. *This valve is structurally normal, it needs not be replaced.* The huge coaptation gap is due to posterior leaflet restriction. A dyskinetic/akinetic wall with apical displacement of the papillary muscle was found in preoperative echocardiographic diagnosis in this patient after a transmural posteromedial myocardial infarction that had occurred several years previously

Fig. 29B.2. Patch enlargement of the restricted posterior leaflet demonstrated here has shown to provide an increase of more than 80% of the surface area. This remodeling technique allows restoring both the normal extent of mitral leaflet coaptation and posterior leaflet motion, which corrects the underlying asymmetric tethering of the papillary muscle to the myocardium. The coaptation gap shown in Fig. 29B.1 is now vanished with a new coaptation surface and abolished mitral regurgitation

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Flexible posterior annuloplasty band (Coogrove-Edwards (R) Annaloplasty System)

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